A Review of Heart Rate Variability Biofeedback (HRVB)Treatment Outcome Studies with an Emphasis on Chronic Pain

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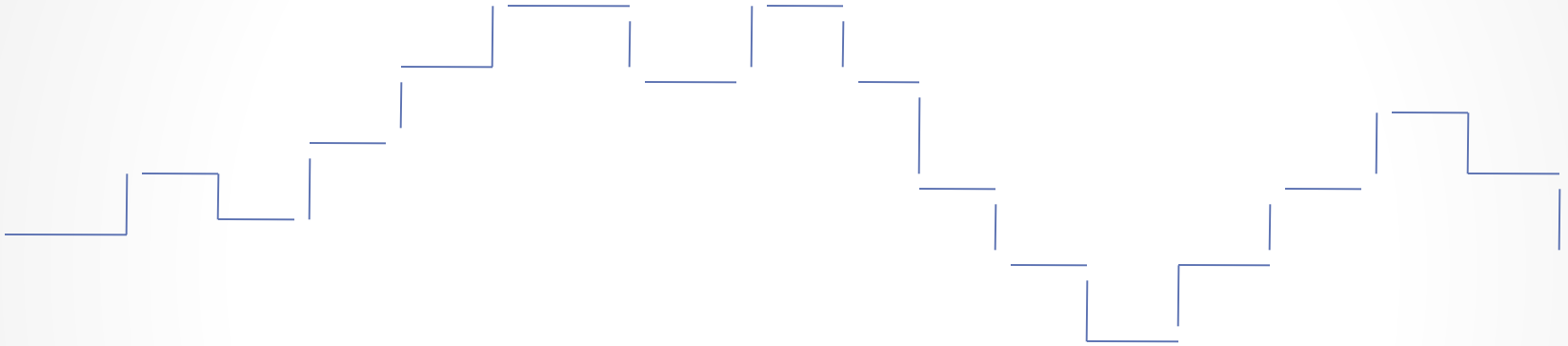
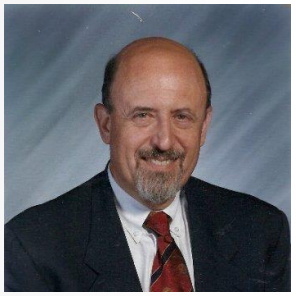
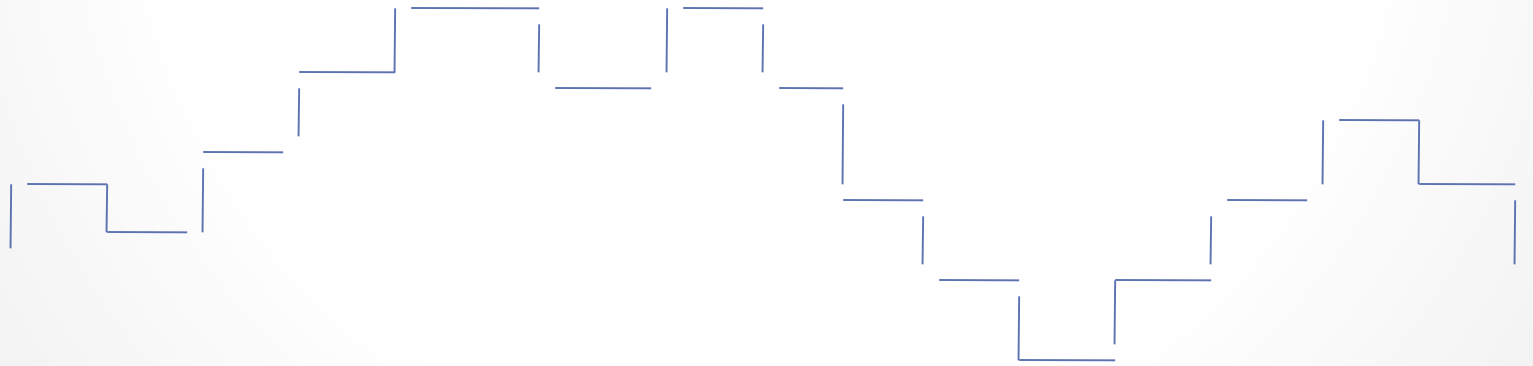


A Very Brief History of HRVB

- Paul Lehrer, Evgeny and Bronya Vaschillo

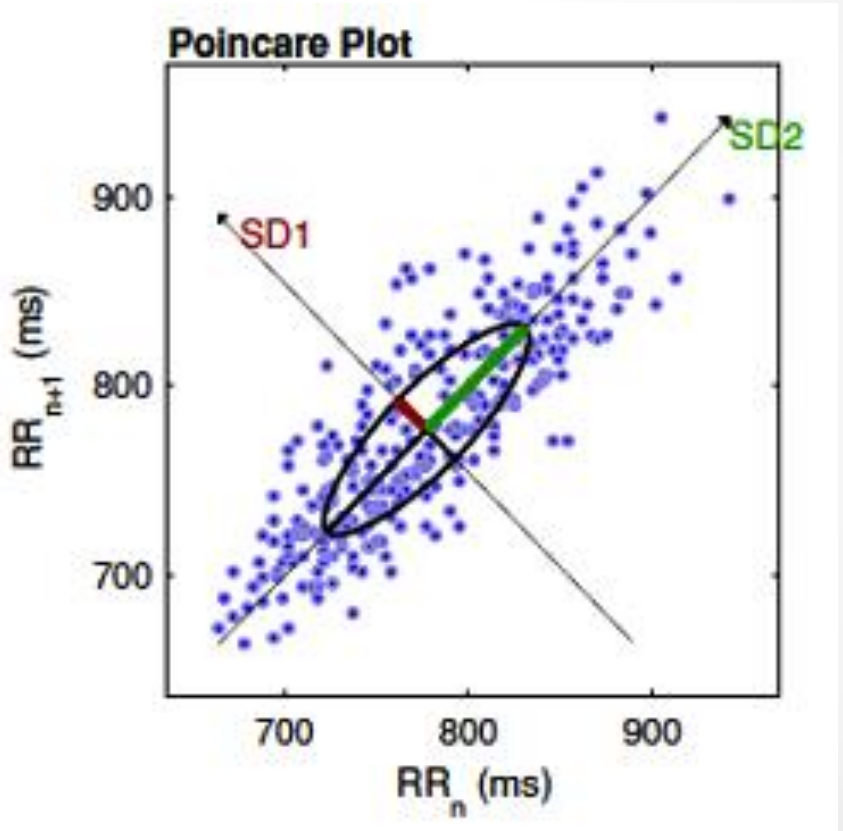


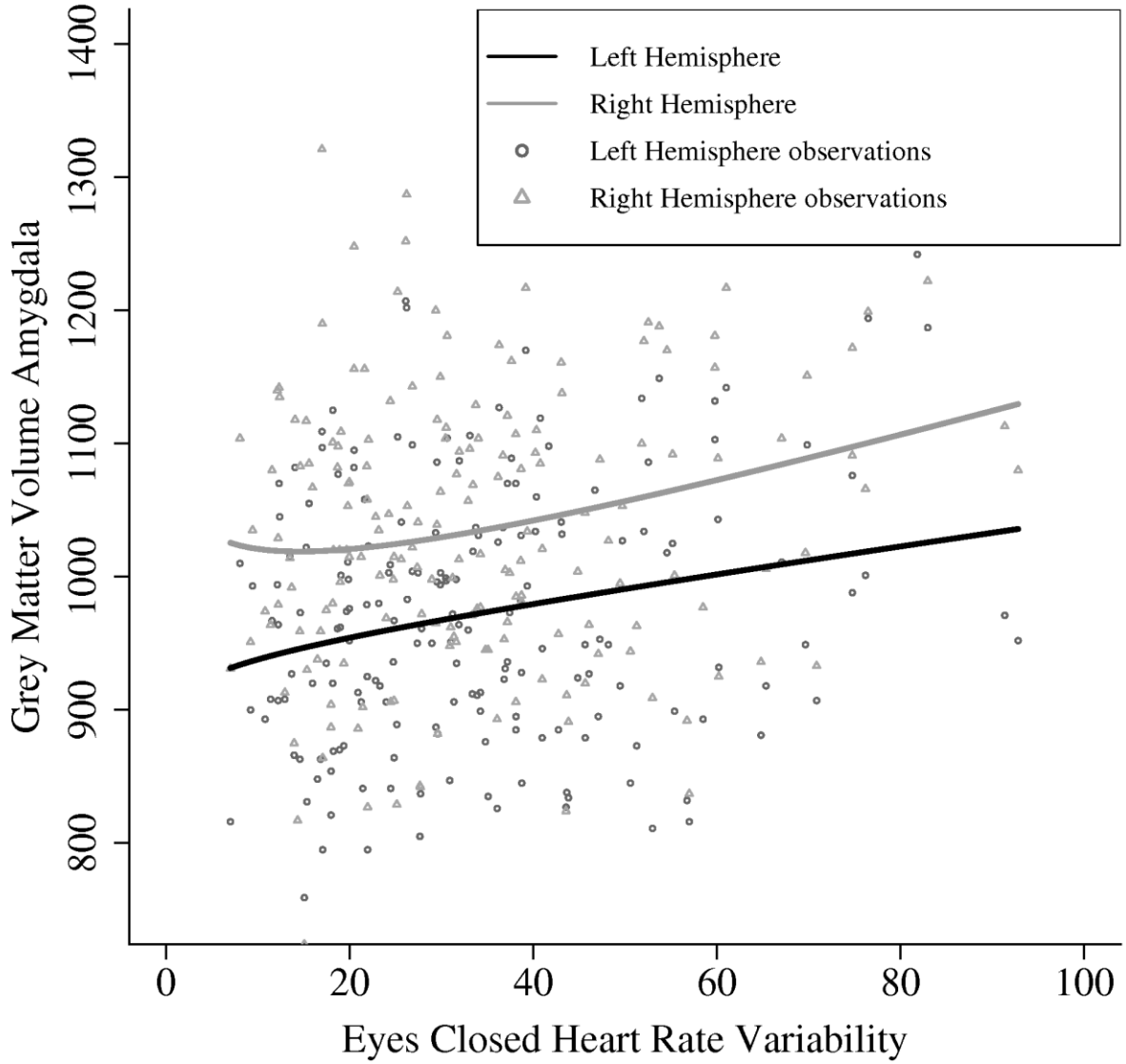



- We were getting mean HRs during exposure, but the data seemed innaproriate for that analysis.
- At the time, we knew little of HRV.

Measurement of HRV as Biomarker Grows Rapidly

- Biomarker for:
  - Cardiac Health
  - Psychological health
  - Emotional regulation
  - other

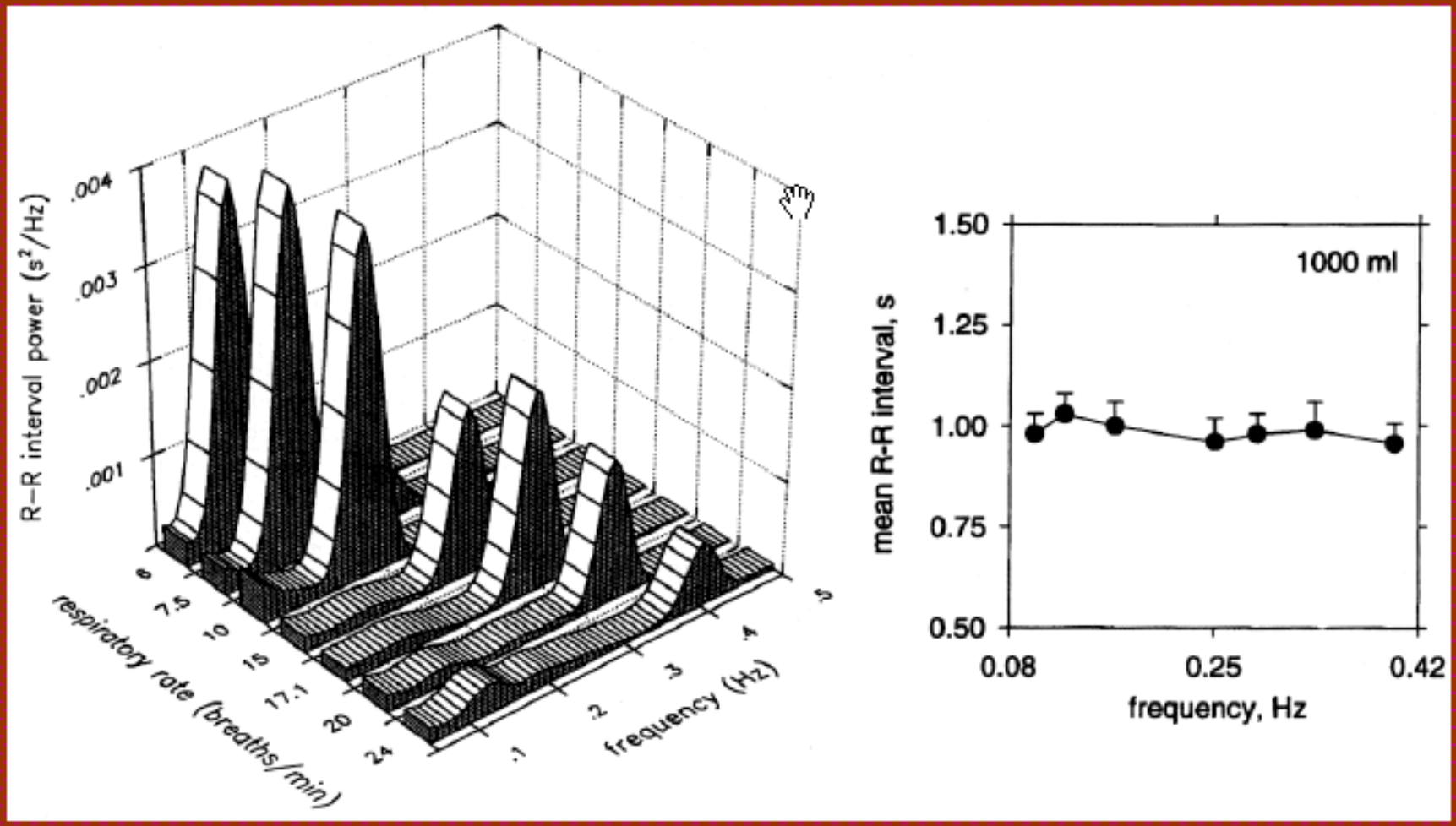




Heart Rate Variability (RMSSD) predicting Amygdala Volume for Left and Right Hemispheres. Increasing Heart Rate Variability is associated with greater amygdala grey matter volume for the Lateral PreFrontal Cortex [LFPC] bilaterally (F = 12.57, R-Squared = 0.122, p<0.0001). Increasing Heart Rate Variability was associated with greater LPFC volume.

1983 with student Diane Herbs

- Our first attempt at HRV or RSA biofeedback
  - Compared to temp training for hypertension
    - Ss able to demonstrate learning quite readily
    - BP reductions comparable to other behavioral studies
  - Formulation of Mediational Model



Average RR-interval spectral power and RR intervals from 10 healthy supine subjects breathing to a nominal tidal volume of 1000 mL at seven breathing rates. From Eckberg DL, Circulation 1997;96:3224 –3232 (originally Brown et al.,1993)

The Afferent pathways

"The brain listening to the heart"

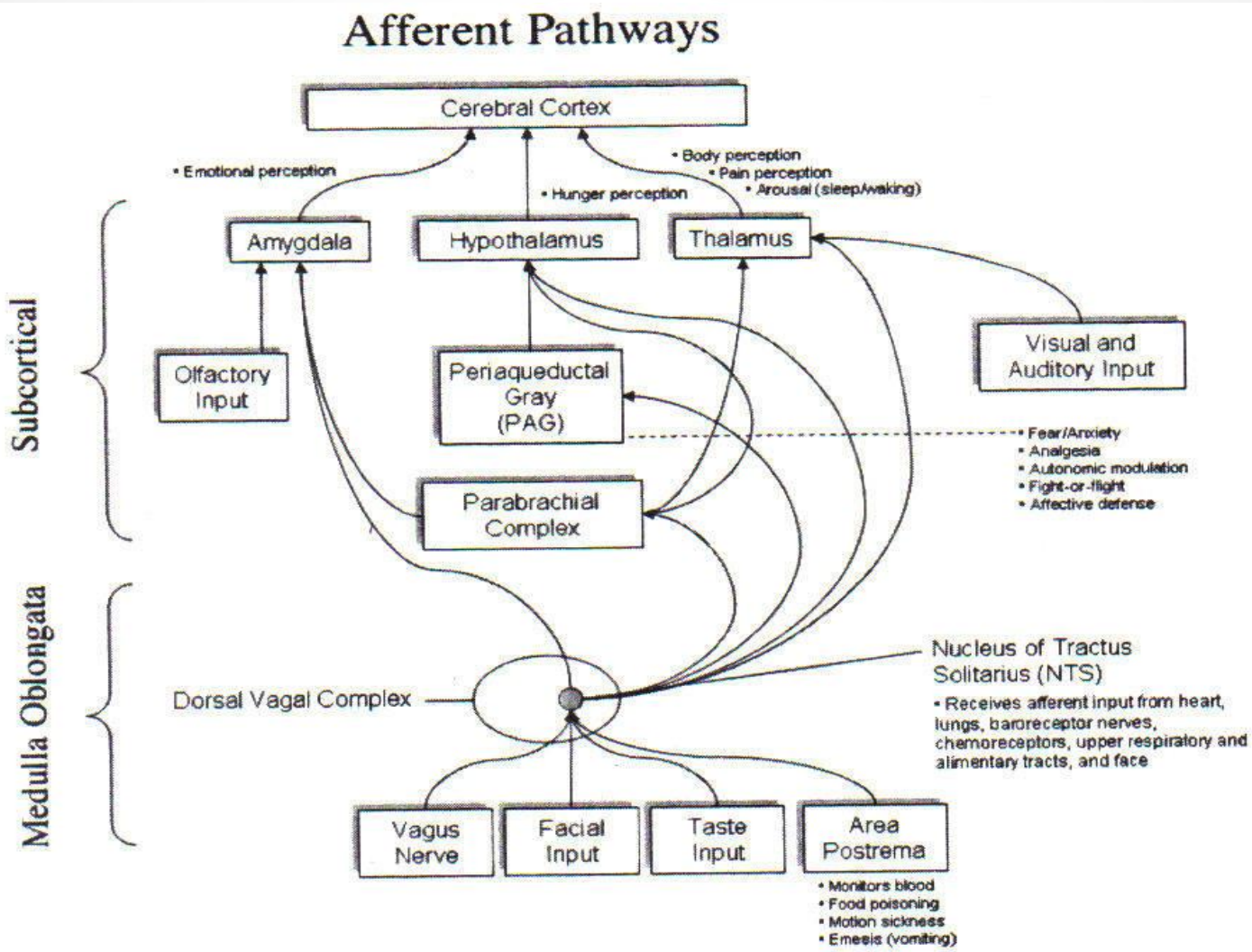
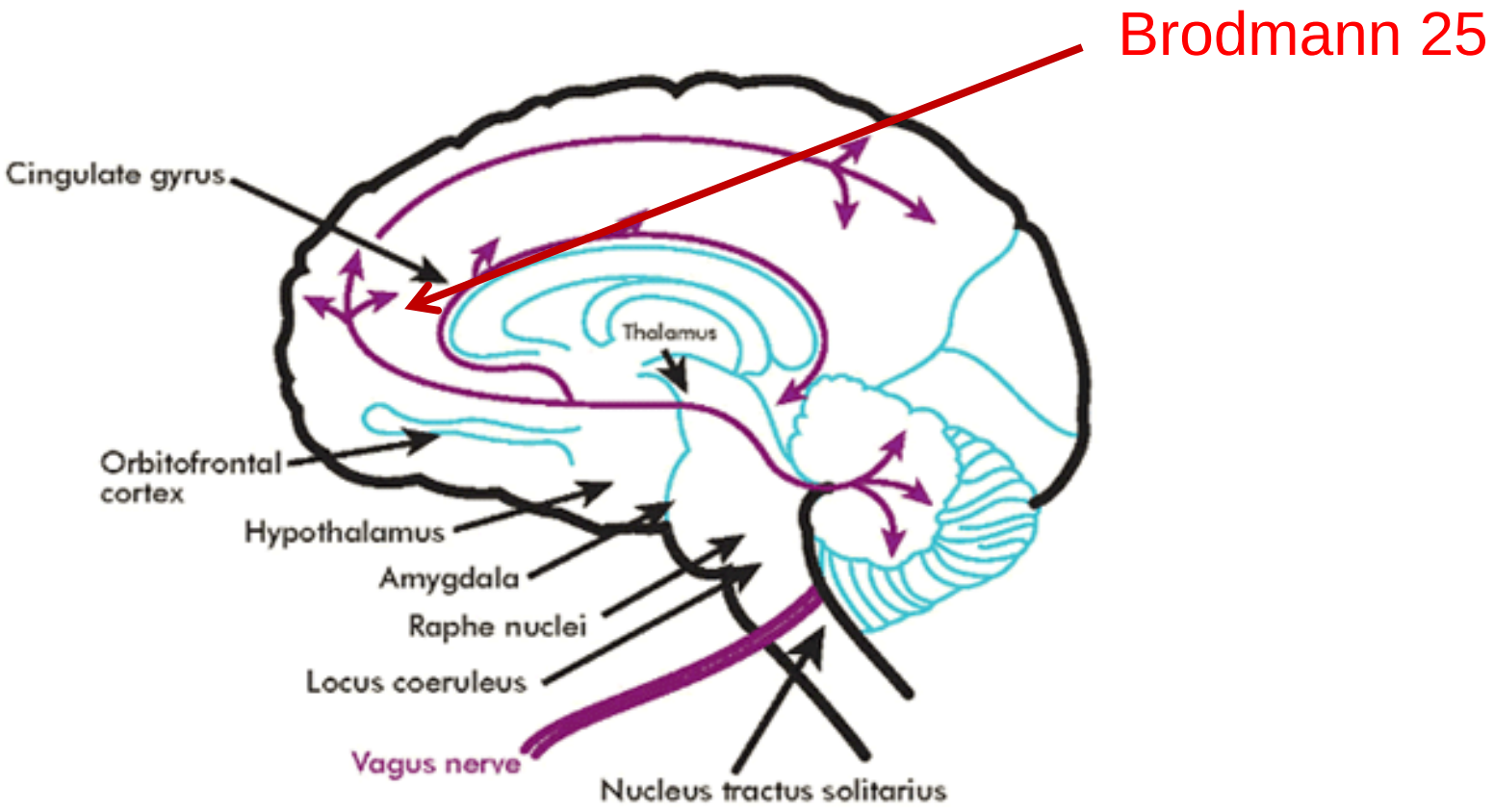
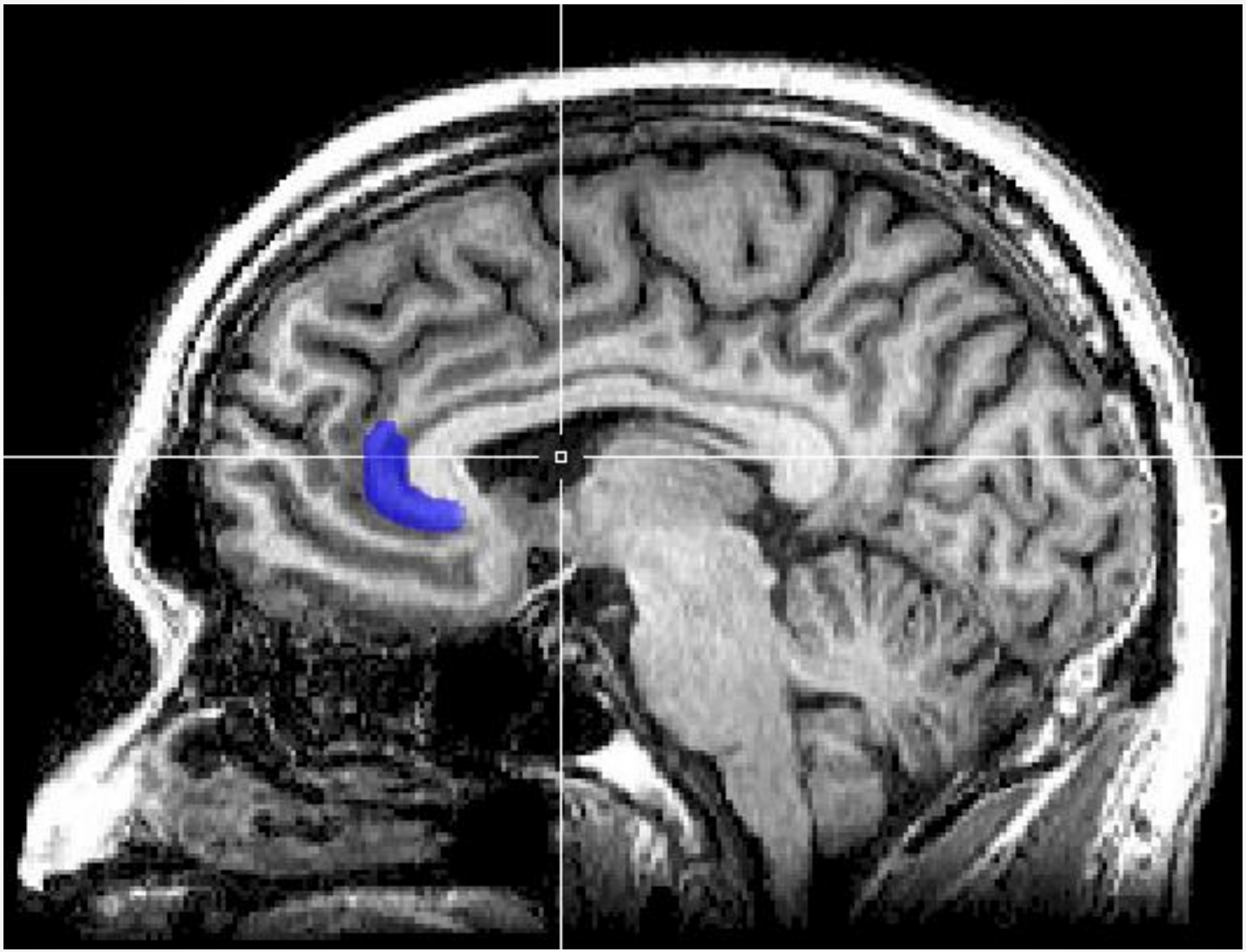


Figure 8. Afferent pathways. Diagram of the currently known afferent pathways by which information from the heart and cardiovascular system modulates brain activity. Note the direct connections from the NTS to the amygdala, hypothalamus, and thalamus. Although not shown, there is also evidence emerging of a pathway from the dorsal vagal complex that travels directly to the frontal cortex.

the familiar pattern, is what underlies the generation of sistent source of dynamic rhythmic patterns in the body.

The vagus nerve: pathway to the limbic system





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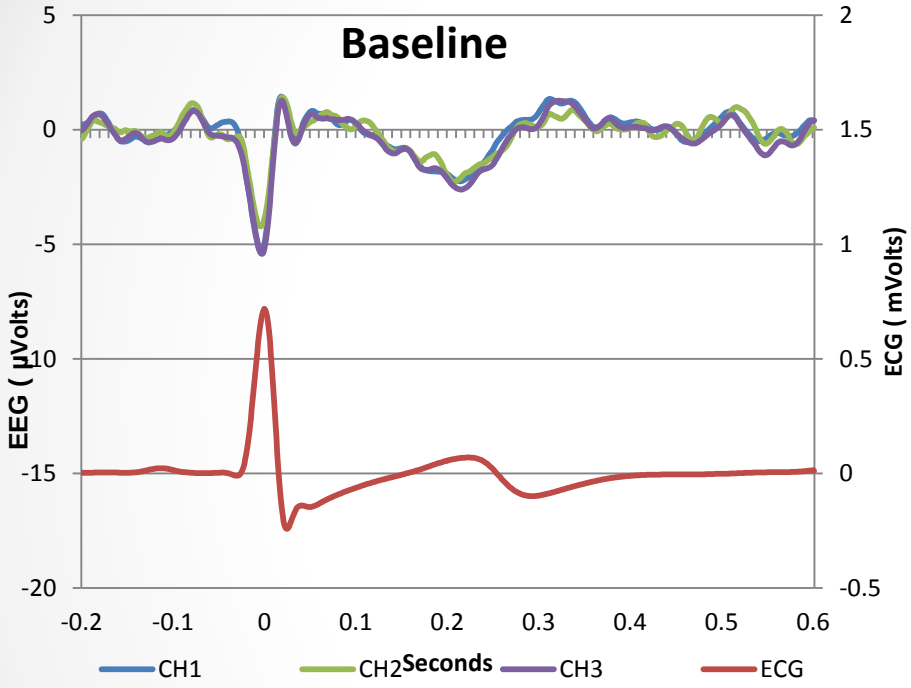
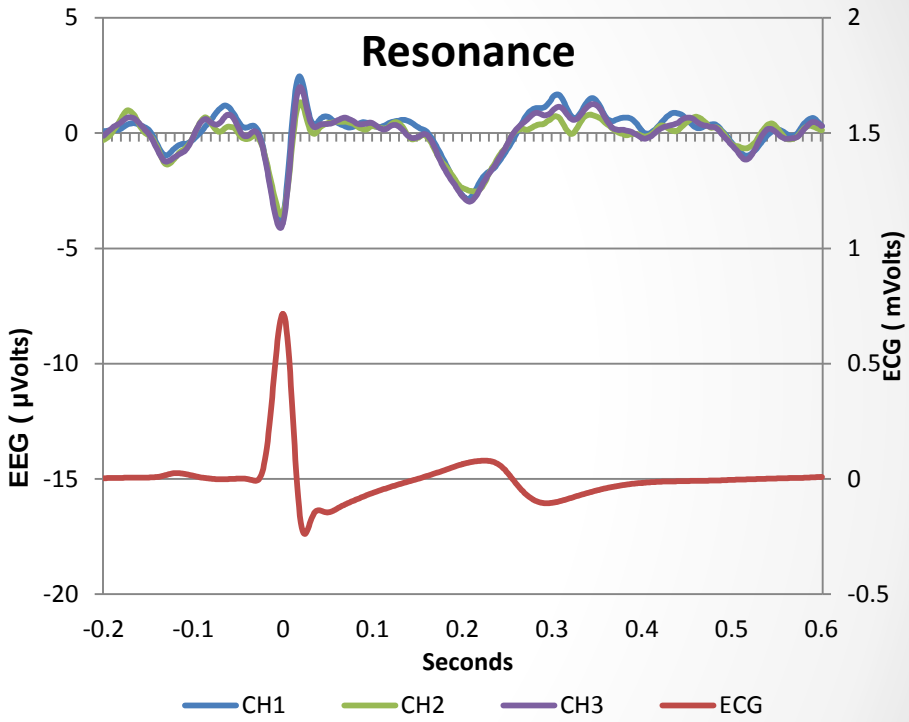
Extinction and the Treatment of Anxiety Disorders

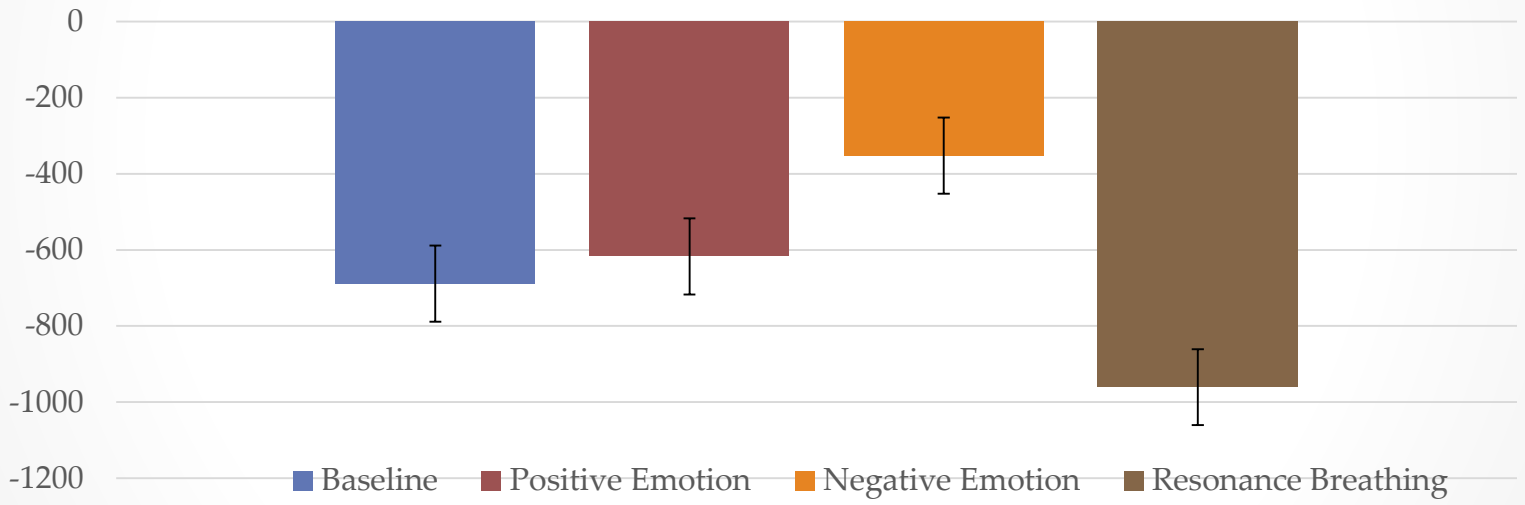
- **Conclusions**
- Extinction paired with VNS is more rapid than extinction paired with sham stimulation. As it is currently approved by the Federal Food and Drug Administration for depression and seizure prevention, VNS is a readily available and promising adjunct to exposure therapy for the treatment of severe anxiety disorders





MacKinnon, S., et al. (2013). "Utilizing heartbeat evoked potentials to identify cardiac regulation of vagal afferents during emotion and resonant breathing." Applied Psychophysiology and Biofeedback **38**(4): 241-255.

Heart Period Evoked Potential Across Conditions



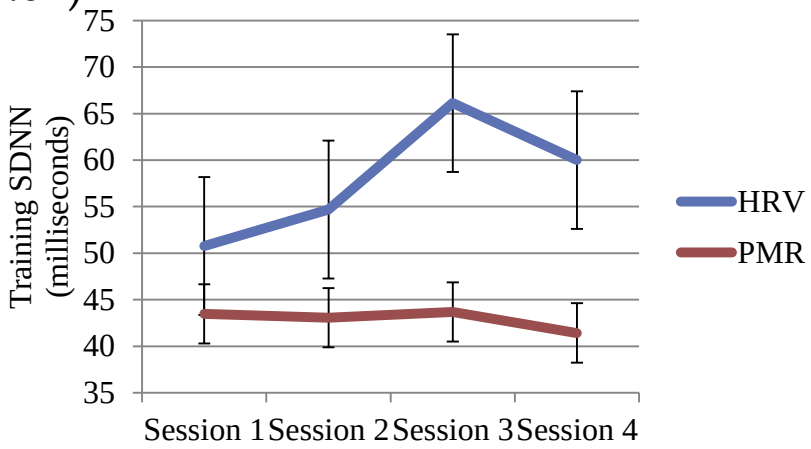
POTENTIAL

Christina Huang, M.A., Ph.D., BCB

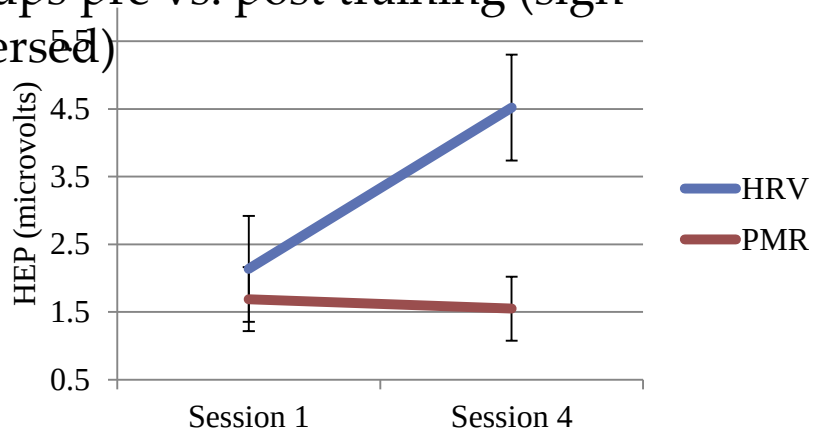
2013)

- Training in HRVB vs EMG Biofeedback Assisted Progressive Muscle Relaxation (PMR) over 4 sessions
- Assess HEP Pre and post training

SDNN between groups over time (p<.01)



HEP at 250 microseconds for both groups pre vs. post training (sign reversed)



Conclusions

- HRVB (probably as an adjunct to CBT or ACT) offers a promising treatment for depressive disorders
- It has the added advantage of improving autonomic homeostasis which has been shown to lead to improved cardiovascular function
- A possible mechanism is emerging
- It may eliminate some of the stigma associated with talk therapies and therefore increase compliance and reduce drop-outs (especially in minorities).
- Much more research is needed with active control groups.

Table 1. Disorders treated with HRVB that are hypothesized to have restoration of autonomic function as the primary mediator

Disorder	Intervention	Design (Control)	Measures	Results	Reference(s)
Asthma					
	HRVB + HT	vs. Sham EEG	Symptoms, lung function, medication	HRVB > control	Lehrer et al., 2000; Lehrer et al., 2004
Chronic Obstructive Pulmonary Disease					
	HRVB + oxymeter fdbk	vs. TAU	6-minute walk	HRVB > TAU	Giardino et al., 2004
Functional Gastrointestinal Disorders					
Recurrent Abdominal Pain	Slow breathing + temp fdbk	vs. TAU	Parent and child symptom ratings	Breathing > control	Humphreys & Gevirtz, 2000
Recurrent Abdominal Pain	HRVB	vs. Control	Symptom ratings and HRV measures	Symptom improvement associated with SDNN gains	Sowder et al., 2010
IBS	HRVB	vs. Hypnosis	IBS symptom severity scale, HADS	Both groups improved equally (HRVB slightly better)	Dobbin, Dobbin, Ross, Graham, & Ford, 2013
Recurrent Abdominal Pain	HRVB integrated into other therapies	Case study	Symptom log	Greatly improved	Masters, 2006
Cyclic Vomiting	HRVB	Case study	Vomiting frequency	Greatly improved	Slutsker, Konichezky, & Gothelf, 2010
Recurrent Abdominal Pain	HRVB	TAU	IBS symptom measures	HRVB > TAU	Ebert, 2013
Fibromyalgia					
Cardiac Rehabilitation	HRVB	vs. TAU	Standard FM scales	BFD > TAU	Hassett et al., 2007
Congestive Heart Failure	HRVB	vs. Sham EEG	6-minute walk	HRVB > sham EEG if LVEF > 31	Swanson et al., 2009
Coronary Artery Disease	HRVB	vs. TAU	HRV measures (SDNN)	HRVB > TAU	Del Pozo, Gevirtz, Scher, & Guarneri, 2004

Table 1. Continued.

Disorder	Intervention	Design (Control)	Measures	Results	Reference(s)
Coronary Artery Disease	HRVB + CBT	HRVB + CBT vs. stress management	HRV measures + adjustment scales	HRVB + CBT > stress management	Nolan et al., 2005
Congestive Heart Failure	HRVB + stress management	Case studies	Harvested heart tissue viability	Training group equal to LVAD	Moravec, 2008; Moravec & McKee, 2013
Hypertension					
	HRVB	vs. Sham EEG	Medication adjustment and BP	HRVB maintained BP with fewer meds	Reinke et al., 2007
Prehypertensives	HRVB	vs. Slow breathing and control, 3 month FU	BP, HRV, BRS	HRVB > either control, improved on BP, HRV, and BRS measures	Lin et al., 2012
Prehypertensives	Slow abdominal breathing + EMG biofeedback	vs. Slow breathing alone	BP, HRV	Slow breathing = EMG feedback > slow breathing alone	Wang et al., 2009
Chronic Muscle Pain					
	HRVB + myofascial release	Four groups: stabilization exercises, HRVB alone, myofascial release alone, or combination	Pain and function measures	Combination superior to other interventions	Gordon & Gevirtz, 2006; Vagades, 2011
	HRVB	Case studies	Trigger point pain	HRVB combined with physical release relieves pain	Gevirtz, 2006

Table 1. Continued.

Disorder	Intervention	Design (Control)	Measures	Results	Reference(s)
	HRVB	vs. TAU	Measures of pain, vitality and social functioning	HRVB > TAU	Hallman, Olsson, von Scheele, Melin, & Lyskov, 2003
OB/Gyn					
Preterm Labor	HRVB	vs. Control sessions	Preterm stress, preterm delivery	HRVB > control for stress 13% vs. 33% preterm delivery (n.s.)	Siepmann et al., in press
PIH	HRVB (StressEraser	vs. Matched case histories	BP, birth weight, gestation length	HRVB > controls for birth weight and gestation length	Cullin et al., in press
PIH	Breathing and temperature	vs. activity management vs. TAU	BP levels logged daily	Biofeedback group halted; rising BPs vs. other groups	Sommers, Gevirtz, Jasin, & Chin, 1989

Disorder	Intervention	Design (Control)	Measures	Results	References
Depression					
	HRVB	No control, single group trial	BDI & Hamilton	Depression reduced markedly	Karavidas et al., 2007
	HRVB with StressEraser + DBT	vs. DBT + relaxation	BDI & Hamilton	HRVB group superior	Zucker, Samuelson, Muench, Greenberg, & Gevirtz, 2009
	HRVB	Depressed vs. healthy control	BDI	Depressed patients reduced on BDI, no changes in controls	Siepmann, Aykac, Unterdorfer, Petrowski, & Mueck-Weymann, 2008
	HRVB	vs. TAU after cardiac surgery	CES-D	HRVB > TAU	Patron et al., 2013
	HRVB	vs. Relaxation	BDI & Hamilton	HRVB > relaxation	Rene, Gevirtz, Muench, & Birkhead, 2011
	HRVB + DBT + Zoloft	vs. Zoloft	BDI & Hamilton	HRVB + Zoloft alone	Rene et al., 2011
Anxiety Disorders					
PTSD	HRVB	vs. TAU	CAPS, trauma symptom checklist	HRVB > TAU	Tan, Dao, Farmer, Sutherland, & Gevirtz, 2011
	HRVB	vs. Control	Information processing	HRVB > information processing	Ginsberg, Berry, & Powell, 2010
	HRVB + DBT	vs. Relaxation	PCL	HRVB = relaxation	Zucker et al., 2009
Phobia	HRVB	Case example	approach phobic object	Improved phobic avoidance	Prigatano, 1972
Anxiety	HRVB	vs. Matched controls	Somatic symptoms	HRVB using HeartMath + control	Nada, 2009
	HRVB	vs. Delayed treatment	Anxiety and mood	HRVB > control	Henriques, Keffer, Abrahamson, & Horst, 2011

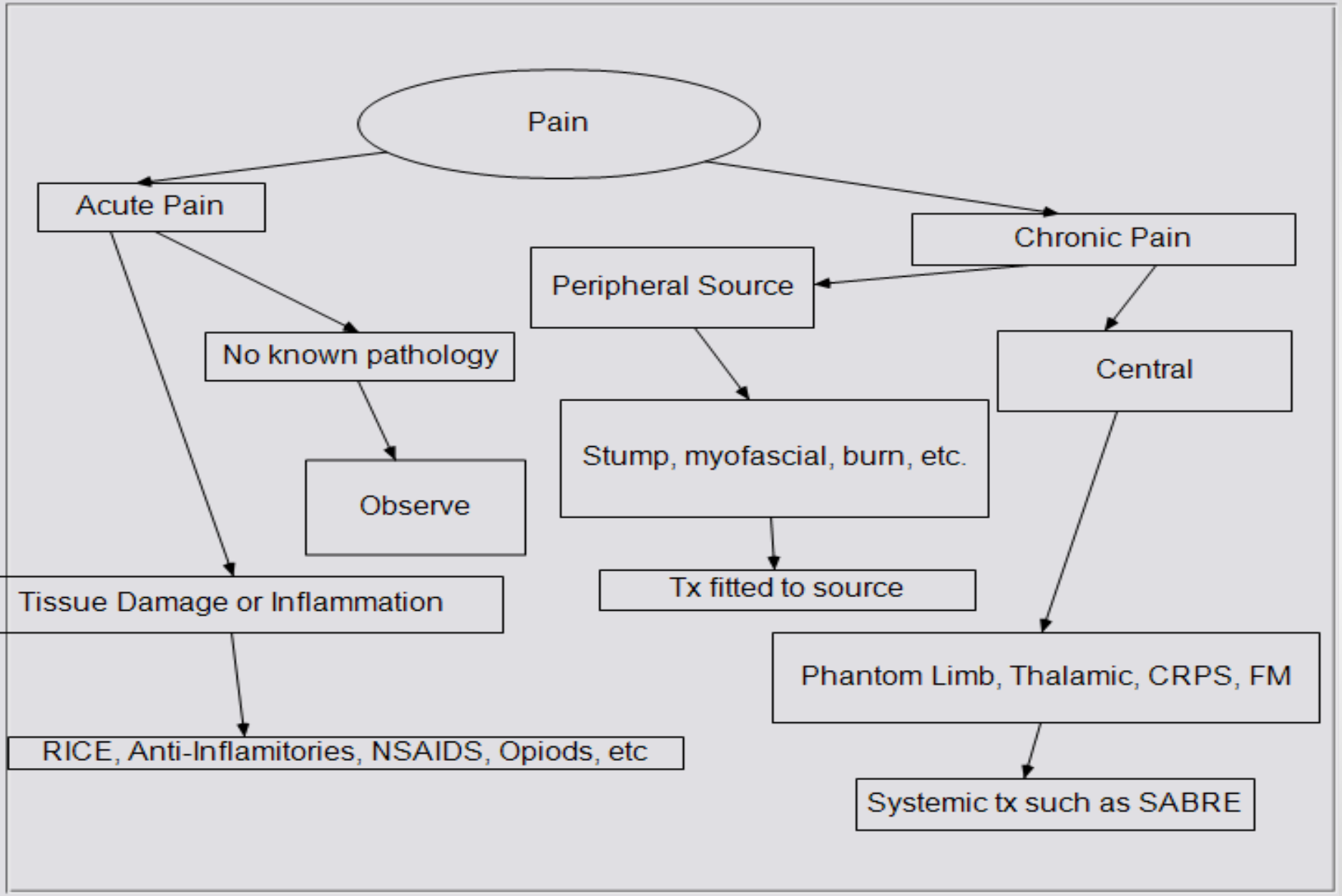
Table 2. Continued.

Disorder	Intervention	Design (Control)	Measures	Results	References
Stress	HRVB + stress management	vs. Control	Cholesterol, glucose, heart rate, blood pressure, positive outlook, and overall psychological distress.	HRVB +> control on all measures, projected cost savings	McCraty, Atkinson, Lipsenthal, & Arguelles, 2009
	HRVB + therapy	Single group study	Anxiety measures	Improvement	Reiner, 2008
Sleep					
	HRVB (StressEraser)	Case report	Sleep log	Insomnia improvement long-term maintenance	McLay & Spira, 2009
Sleep Lab Insomnia	HRVB	vs. Control	Sleep disturbance scale + actigraphy	HRVB > controls	Ebben et al., 2009
Performance					
Baseball	HRVB	vs. Sports psychology control	Hitting performance	HRVB > controls	Strack & Gevirtz, 2011
Golf	HRVB	Case study	Golf performance	Reduced anxiety, improved performance	Lagos, Vaschillo, Vaschillo, Lehrer, & Bates, 2008
Dance	HRVB	vs. Neurofeedback vs. control	Refereed dance ratings	HRVB and neurofeedback > control	Raymond, Sajid, Parkinson, & Gruzelier, 2005
Dance	HRVB	vs. Neurofeedback vs. control	Refereed dance ratings	No effect on dance HRVB reduced anxiety	Gruzelier, Thompson, Brandt, & Steffert, in press
Music	HRVB (emWave)	vs. Control	Performance anxiety measures	HRVB > control	Thurber, 2006
	HRVB or slow breathing	vs. Control	State anxiety	HRVB and slow breathing > control	Wells, Outhred, Heathers, Quintana, & Kemp, 2012

Disorder	Intervention	Design	Measure	Results	Reference
Post-Partum Depression	HRVB (stress eraser)	Vs. TAU, (but random assignment)	Edinburgh Post Natal Depression Scale(EPND)	HRVB had less Anx., sleep disturbance at 1 month than controls	Kodama et al, 2014
	HRVB (Em Wave)	pilot	STAI, EPND	Dep reduced	Beckham&Meltzer-Brody, 2013
Sleep	HRVB	vs. Autogenic vs. control	HF amplitude during sleep	HRVB> AT> Control	Sakakibara et al, 2013
Cardiac Rehab-CAD	HRVB	vs. WLC	SDNN, lnLF, lnHF, hostility	HRVB – SDNN> WLC; Hostlity < in HRVB vs. WLC	Lin, Fan et al., 2015
BP	Slow breathing @6/min	vs. music 10/min breathing	BP, BRS, HF	Slow breathing group <BP, >HF, >SDNN, >BRS	Pietro Amedeo Modesti • Antonella Ferrari • Cristina Bazzini • Maria Boddi, 2015

Pain Classification

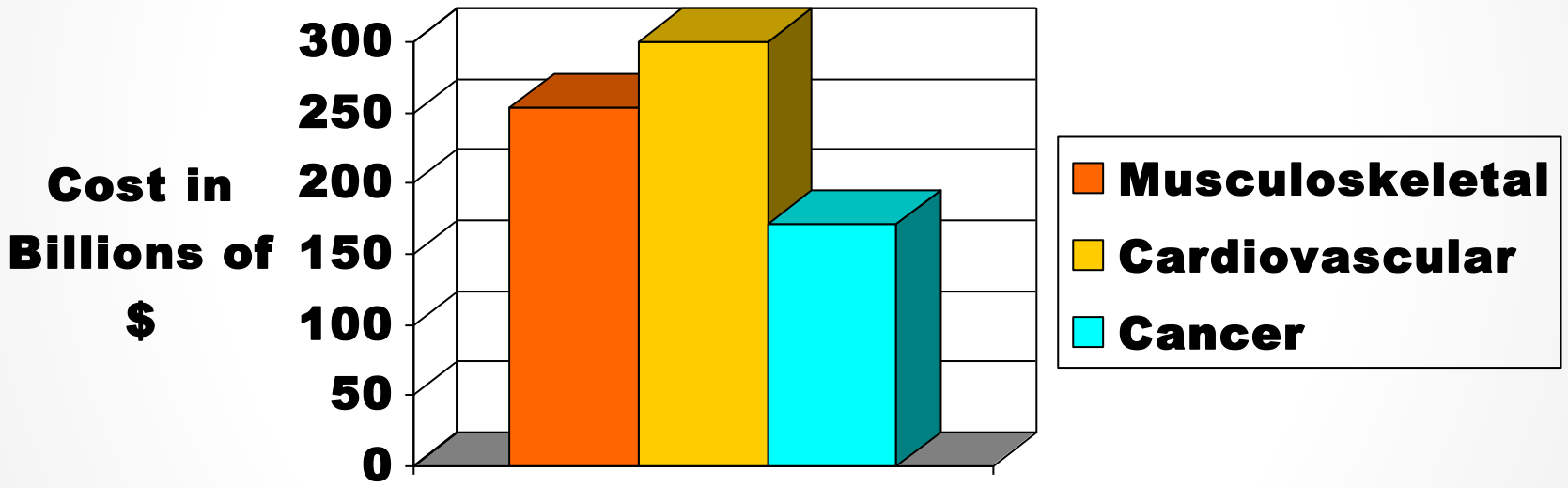
A critical step in treatment


Pain
Acute Pain
Chronic Pain
Peripheral Source
No known pathology
Central
Stump, myofascial, burn, etc.
Observe
Tissue Damage or Inflammation
Tx fitted to source
Phantom Limb, Thalamic, CRPS, FM
RICE, Anti-Inflamitories, NSAIDS, Opiods, etc
Systemic tx such as SABRE

Collaborators

- David Hubbard, M.D. & Myopoint
- Greg Berkoff, D.C.
- Sonja Banks, Ph.D.
- Carol Lewis, Ph.D.
- Walt McNulty, Ph.D.
- Toni Cafaro, Ph.D.
- Jeri Muse, Ph.D.
- Janeen Armm, Ph.D.
- Ali Oliviera, M.S., R.N.
- Jan Vagedes, M.D.
- ChristopherGordon, PT
- Frank Andrasik, Ph.D.

Comparative Costs of Musculoskeletal, Cardiovascular, & Cancer to the U.S. Economy



Chronic Muscle Pain-Epidemiology

- Second only to common cold for medical treatment
- 75-80 million people seeking treatment(Bonica,1992)
- 550 million workdays lost
- $147 billion dollars lost in direct and indirect costs (Am. Assoc. Orthopedics, 1995)
- $**245 billion 2001 dollars**
- 70% of workers compensation claims
- 35% of work-disabling injuries (Calif. Work comp Bull,1991)

Central vs. Peripheral Pain:

A key differential diagnostic distinction

- It is necessary to form a hypothesis on the source or sources of pain
  - Central
    - FM- Central allodynia or sensitization
    - CRPS- pain in a limb
    - Phantom limb pain
  - Peripheral
    - Myofascial Pain
    - IBS
    - Neuropathic Pain
  - Combination

Epidemiology-continued

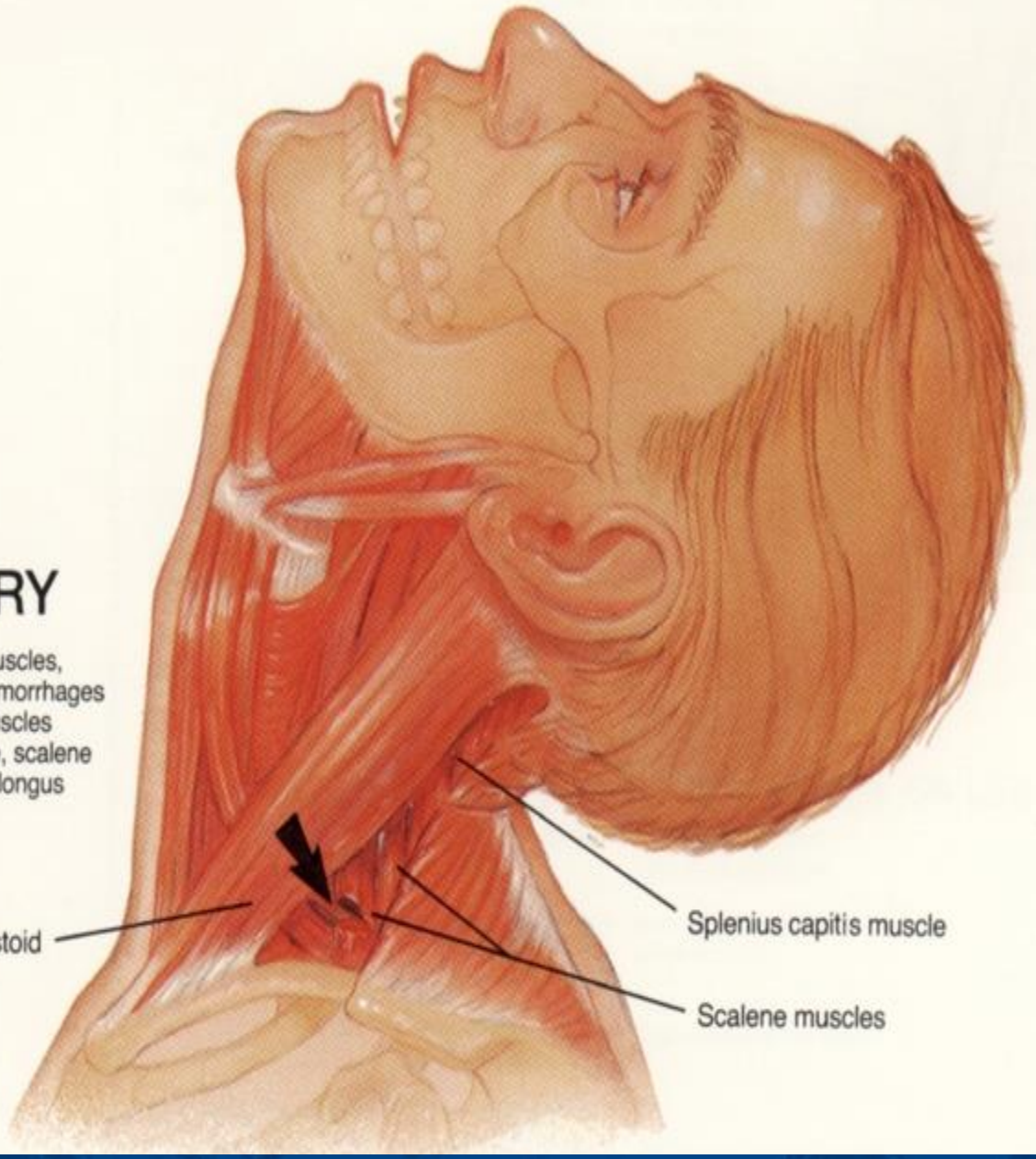
- 45 million ER visits per year (Swiontkowski & Chapman, 1995)
- 70 million physician visits/year (Hollbrook, 1991)
- 425 million visits to chiropractors and "alternative" providers , $4.0 billion(Eisenberg, 1993)
- 20% of general population(Magni,1993)
- 80% lifetime incidence (Bonica, 1990)

Oliveira, Gevirtz, & Hubbard (2005), *Spine*

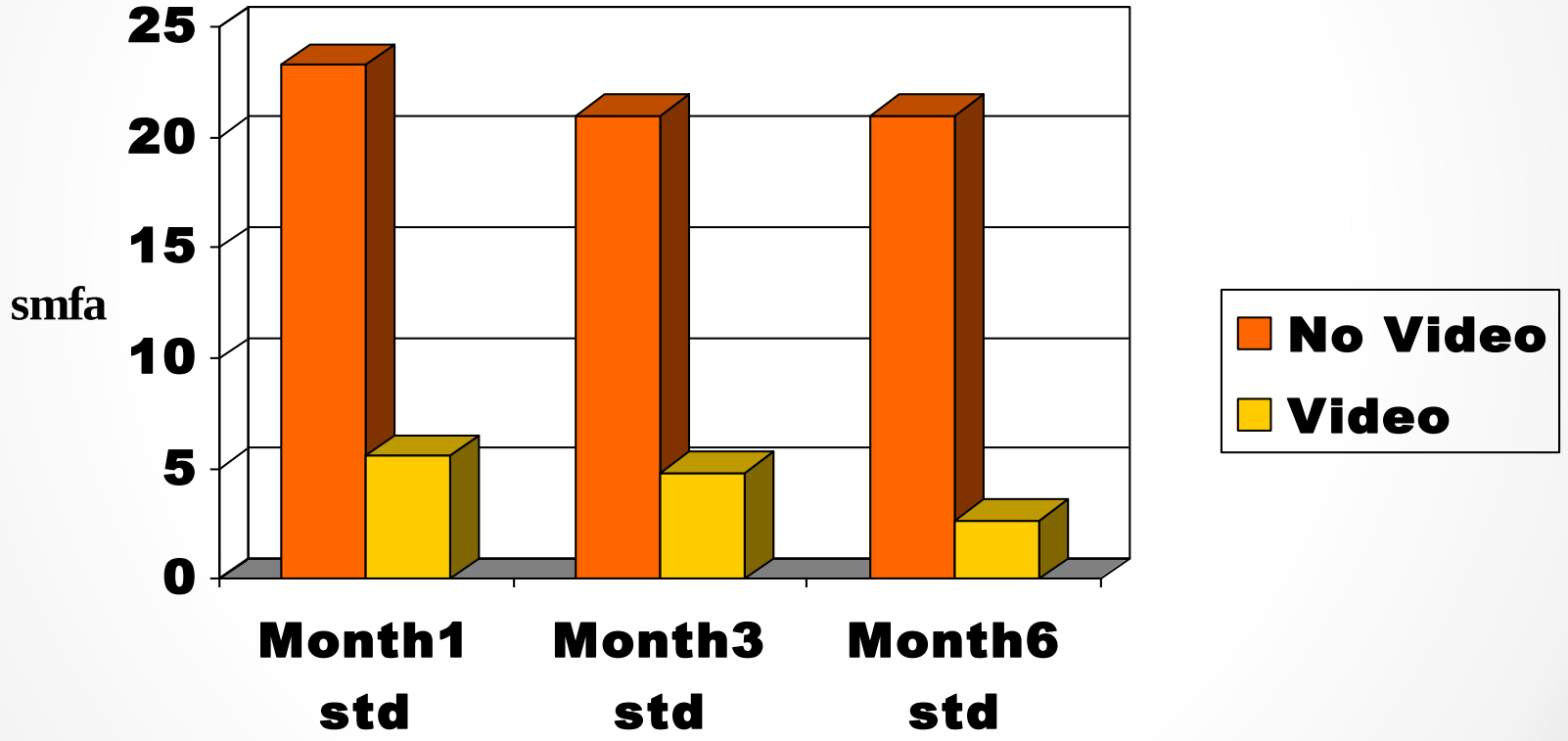
- 126 Whiplash pts randomly assigned to video or normal ER tx
- Groups well matched
- Followed at 1,3 & 6 months
- Video group(as compared to controls) showed good mastery of a content test on TPs ($F(_{1,124})=262.2$, $Eta^2=.9$)
- All ANOVAs and Chi^2 sig., $p<.001$

MUSCLE INJURY

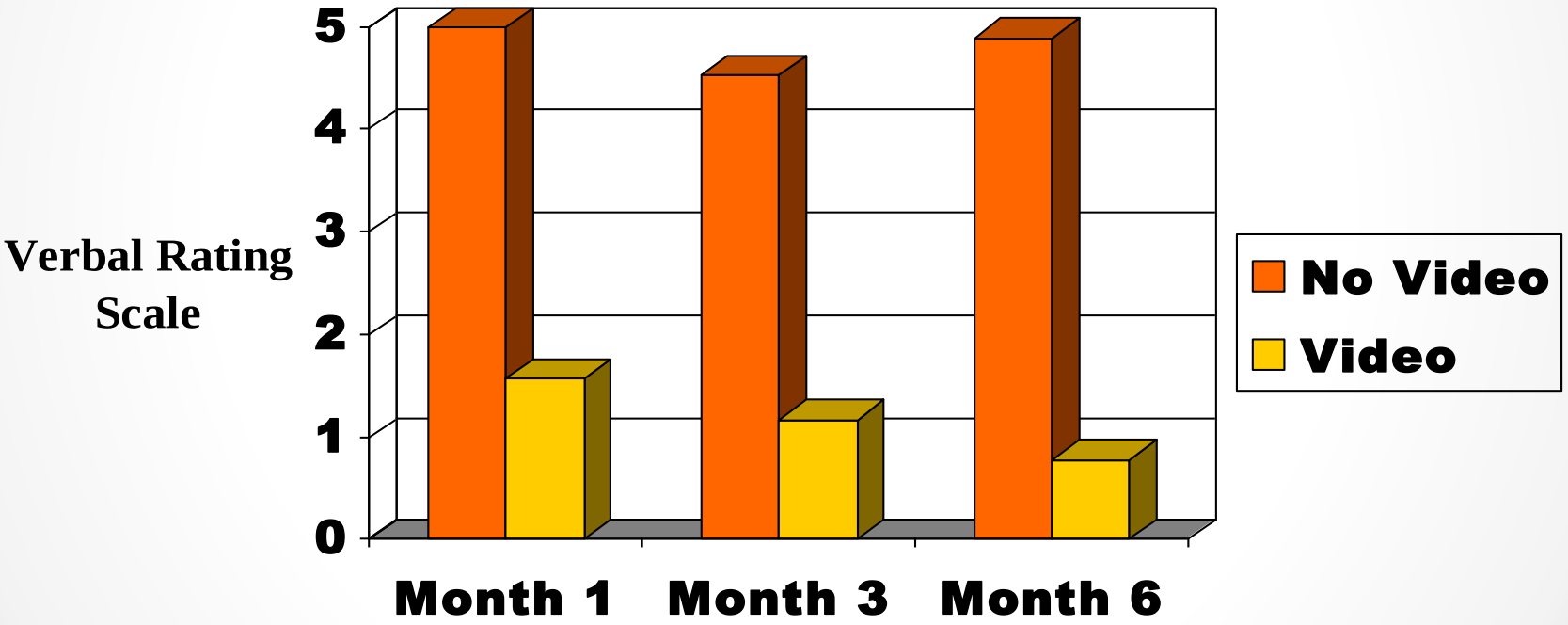
Whiplash can cause injuries of neck muscles, ranging from minor strains and microhemorrhages to severe tears. Commonly affected muscles include the sternocleidomastoid muscle, scalene muscles, splenius capitis muscle, and longus colli muscle.



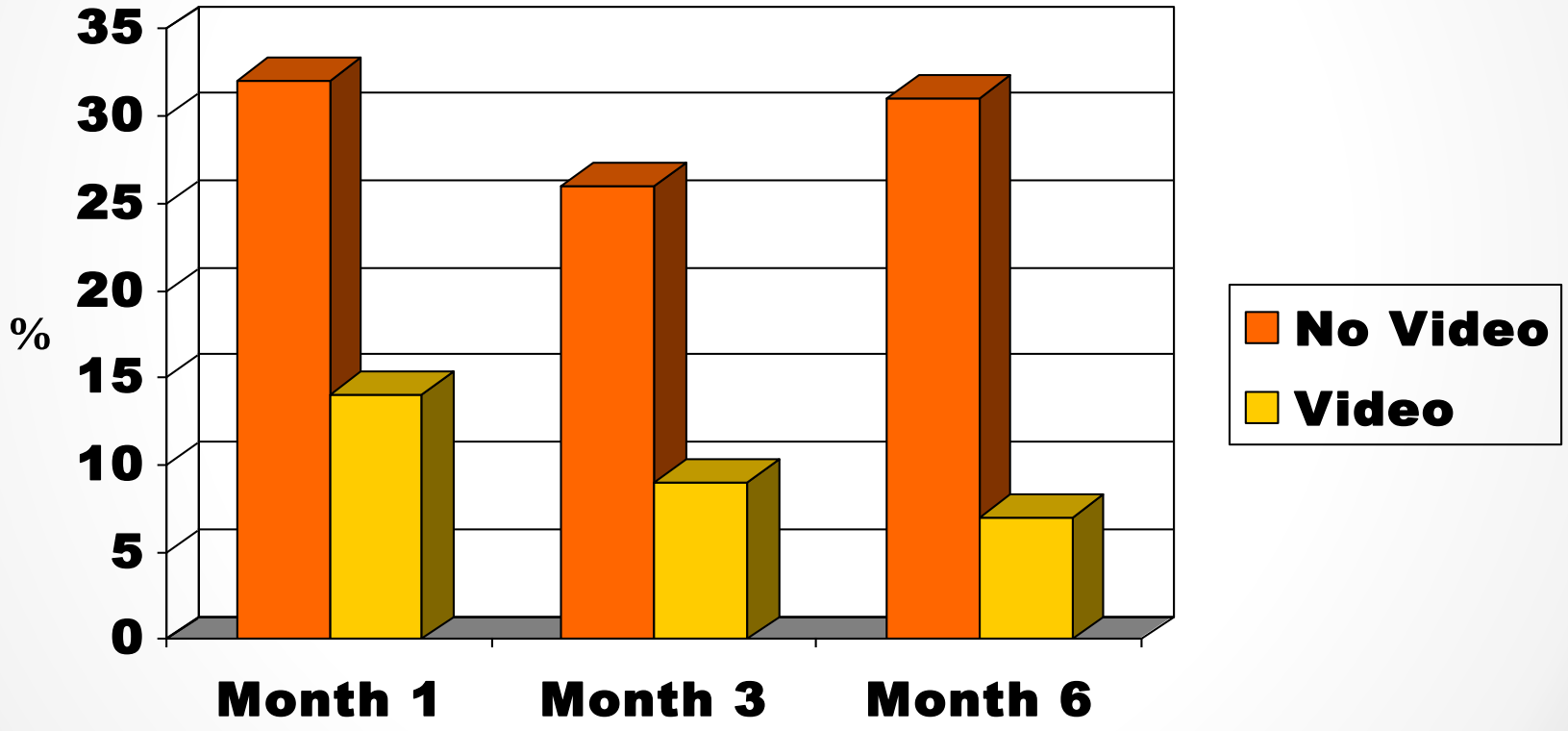
Short Form Musculoskeletal Function Assessment



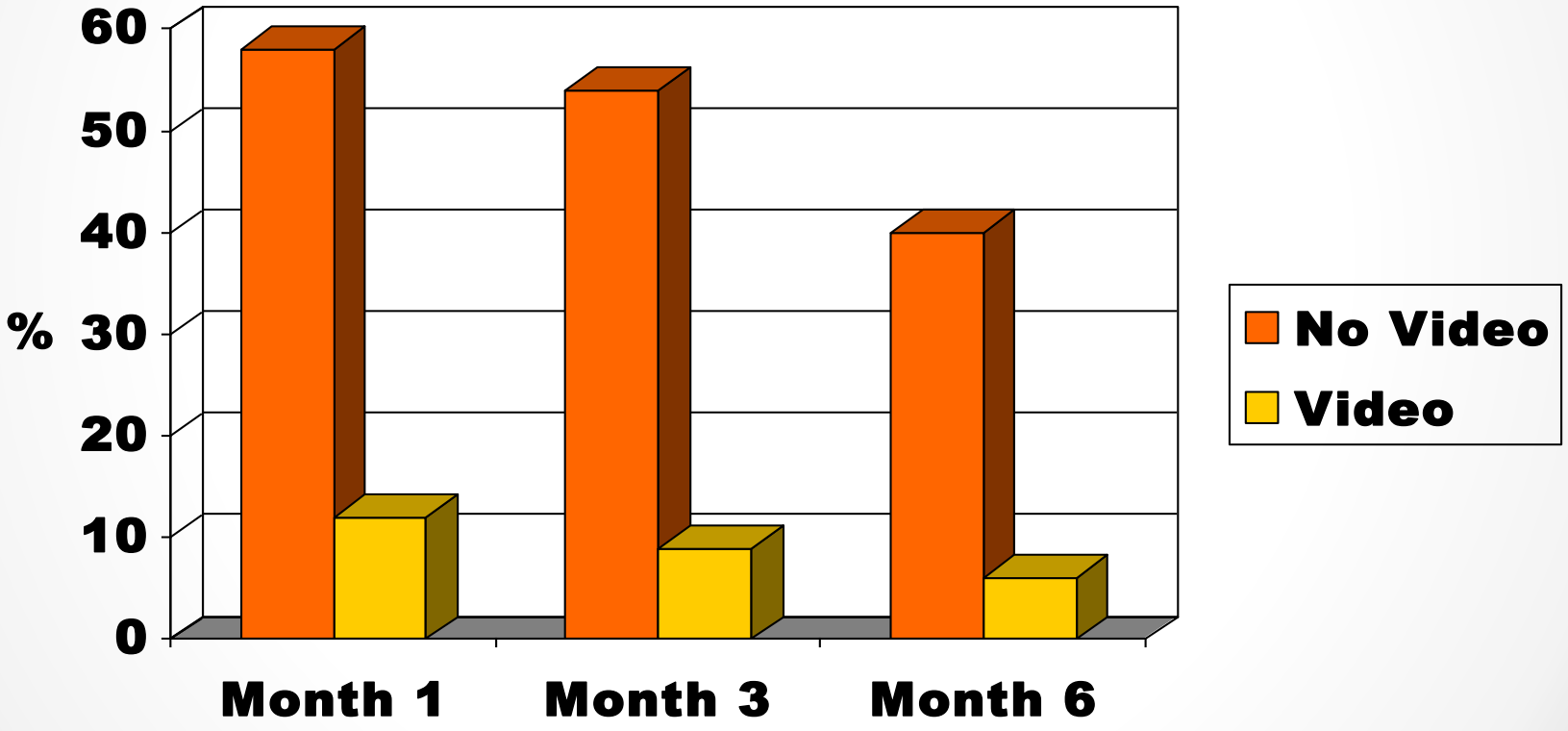
Verbal Rating Scale



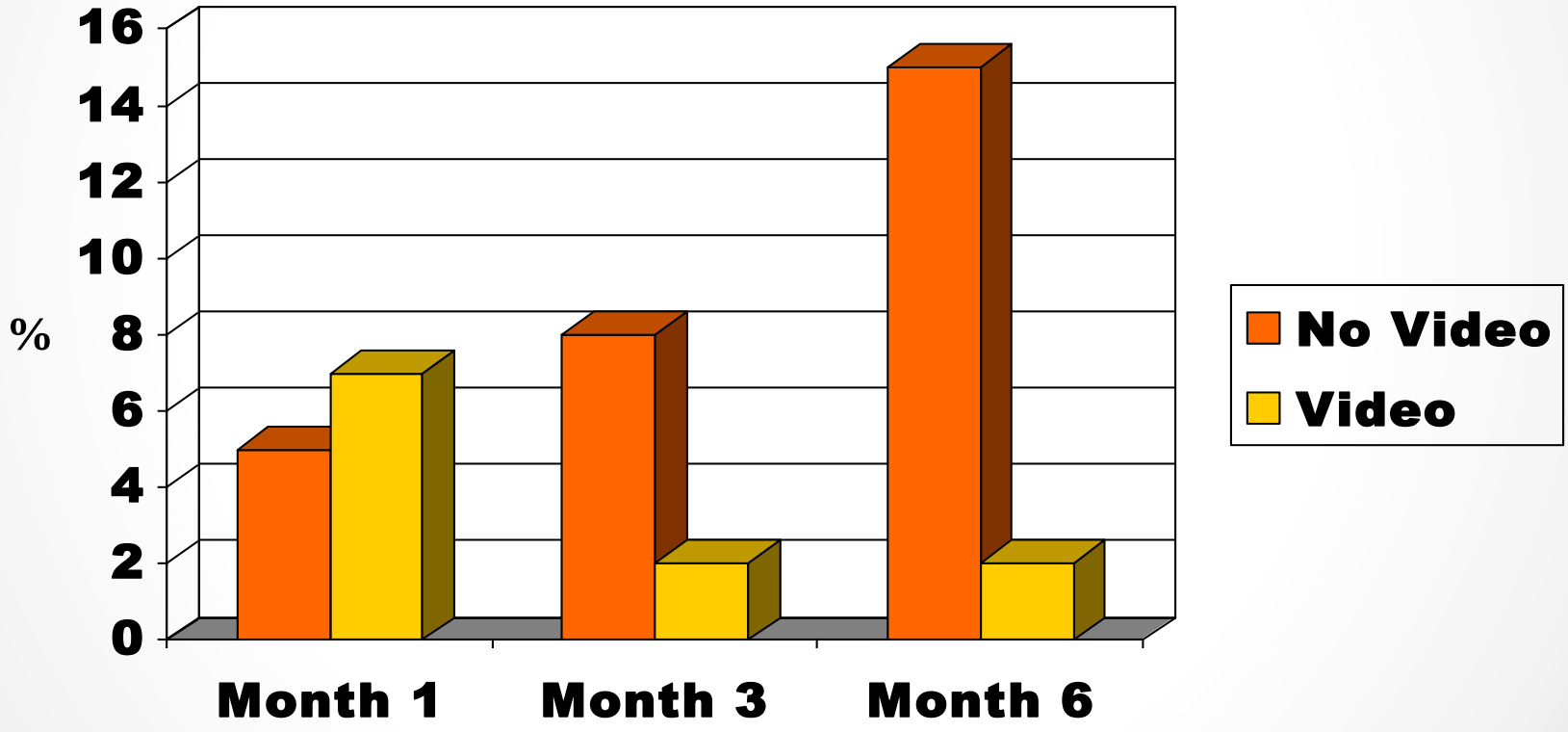
Chiropractic Visits



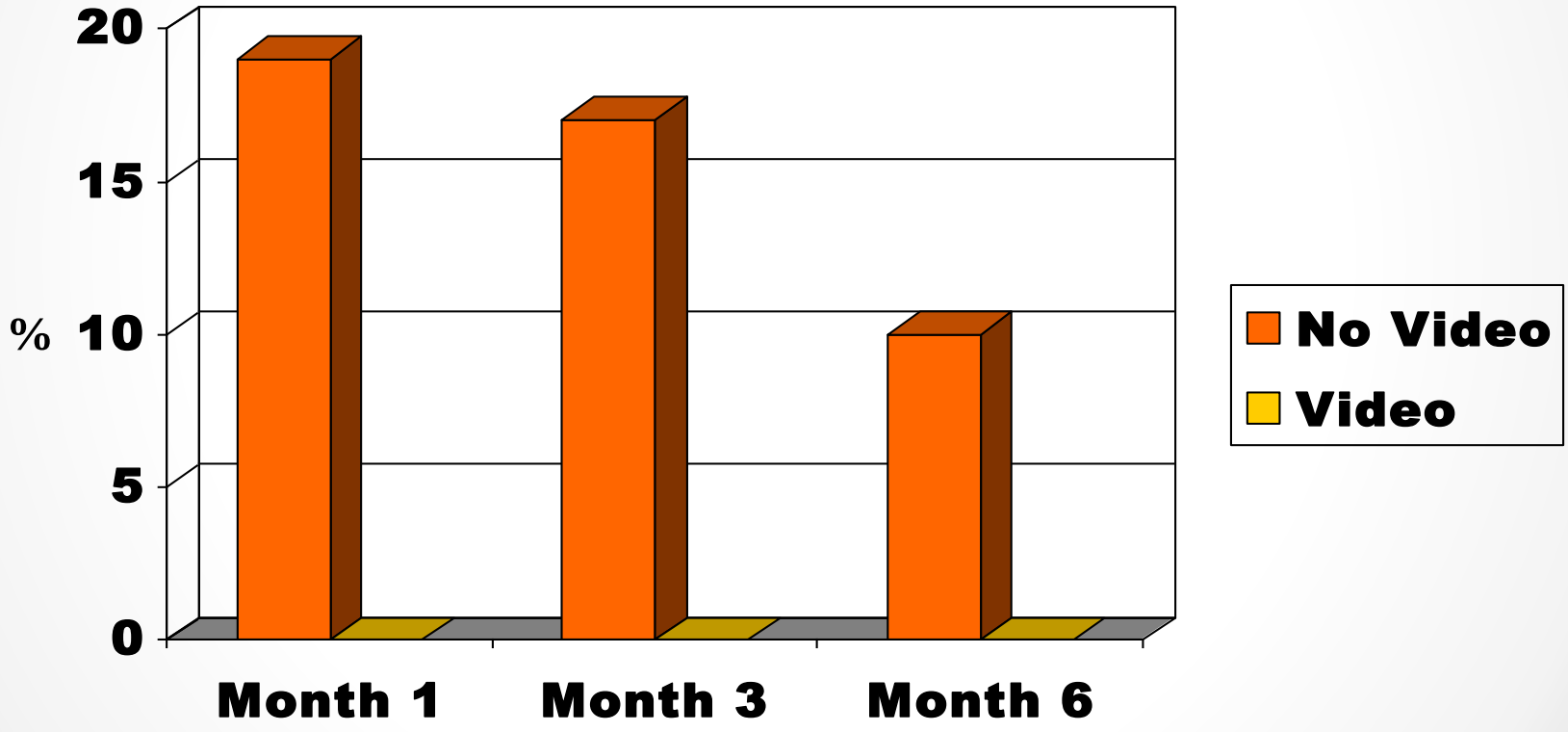
Physical Therapy Visits



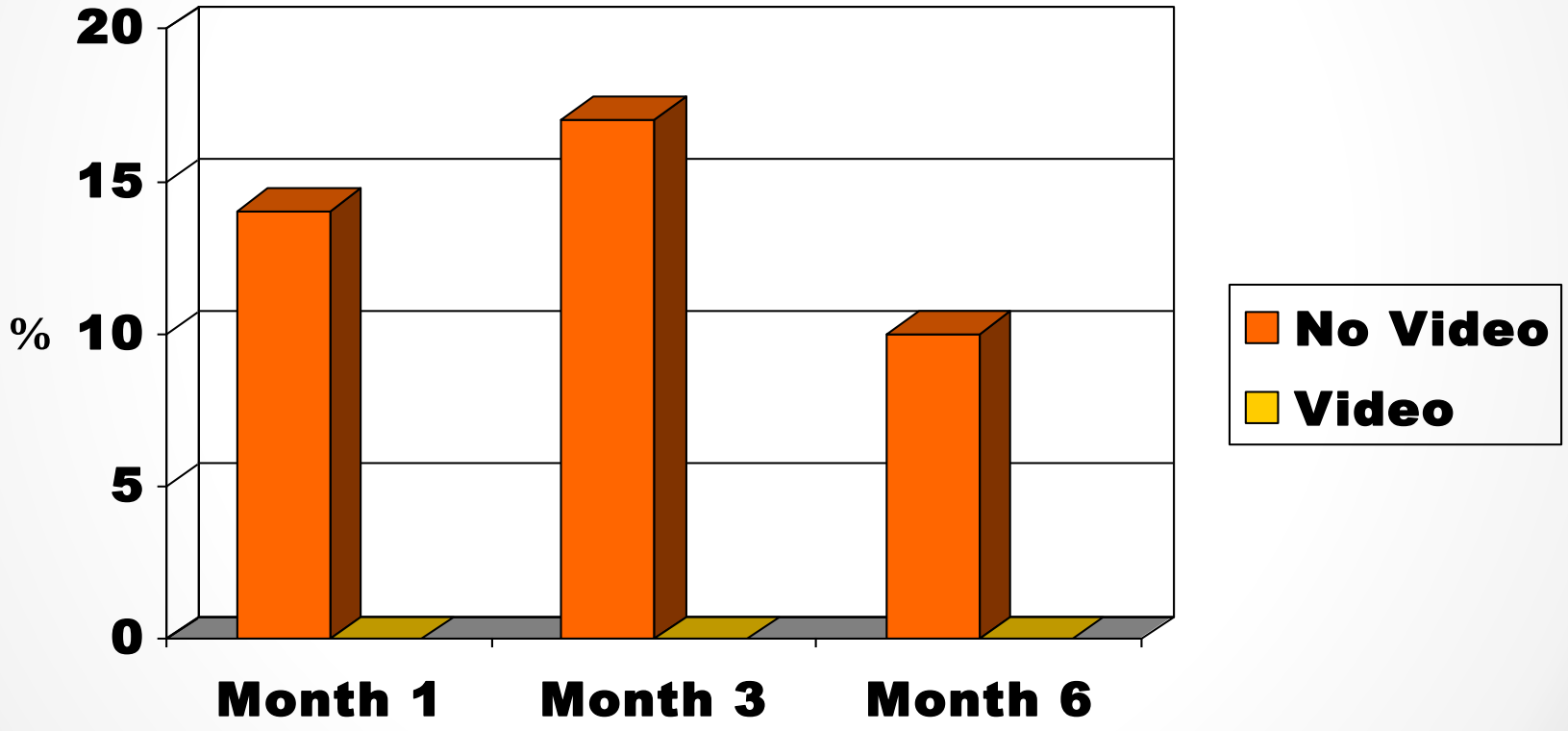
MRIs



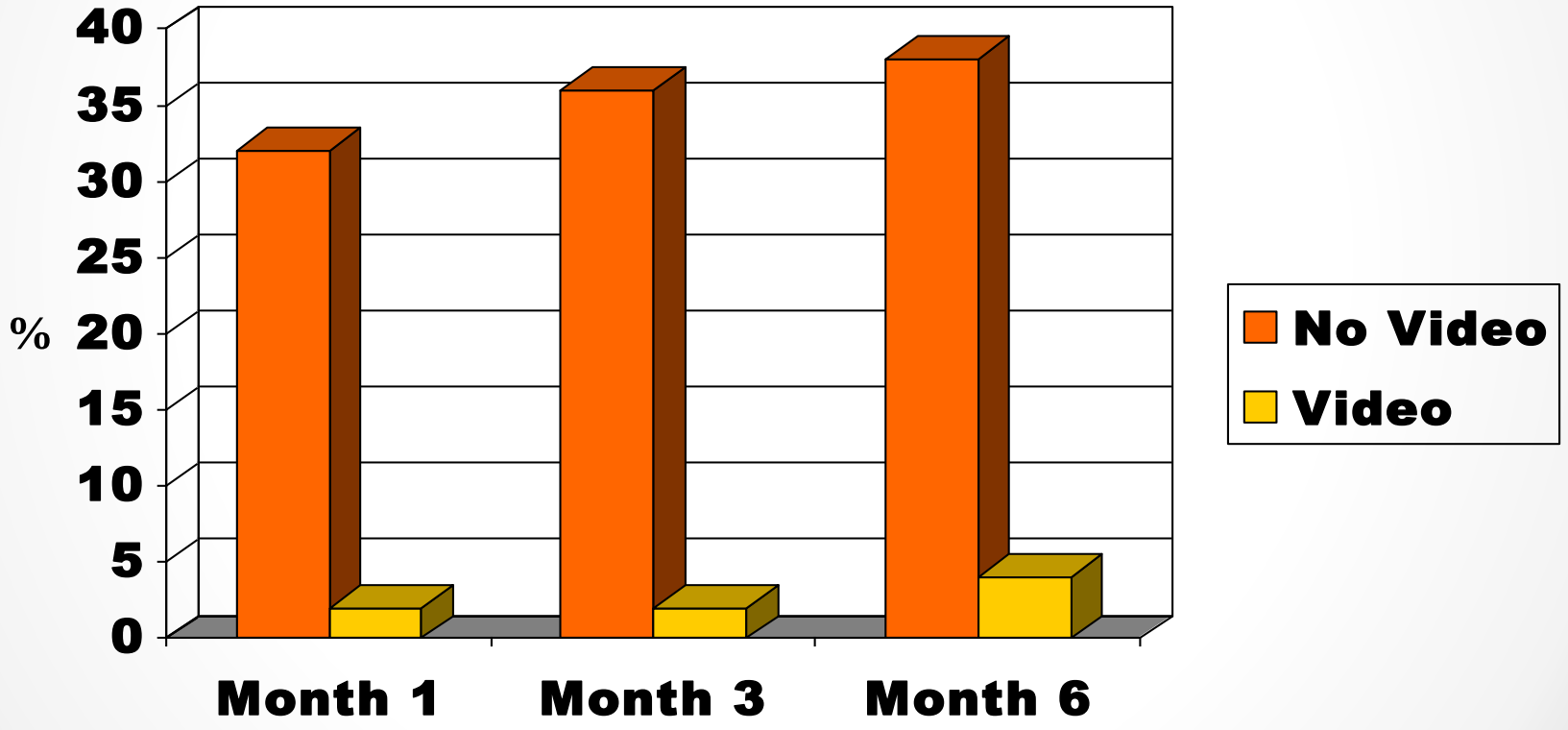
ER Visits



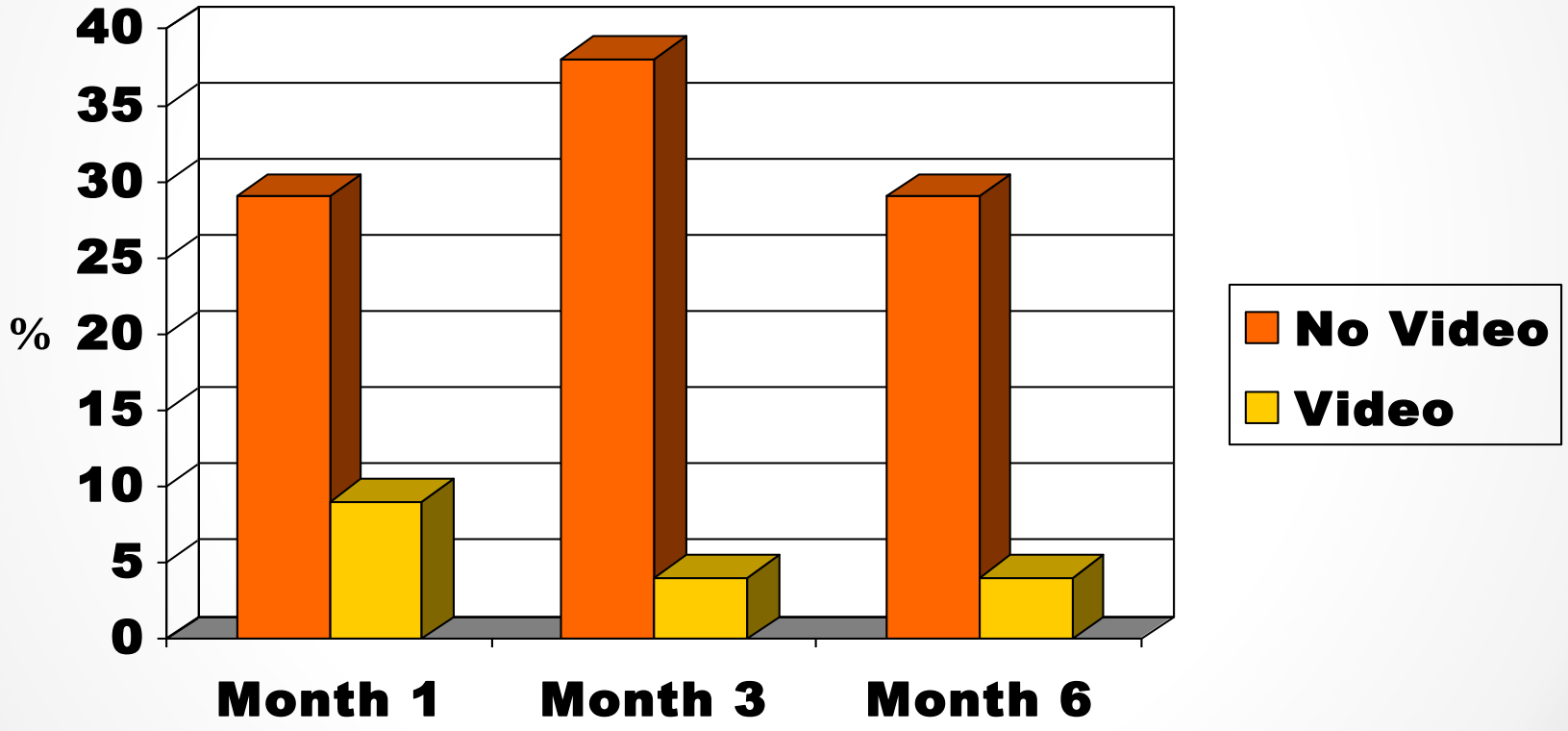
Urgent Care Visits



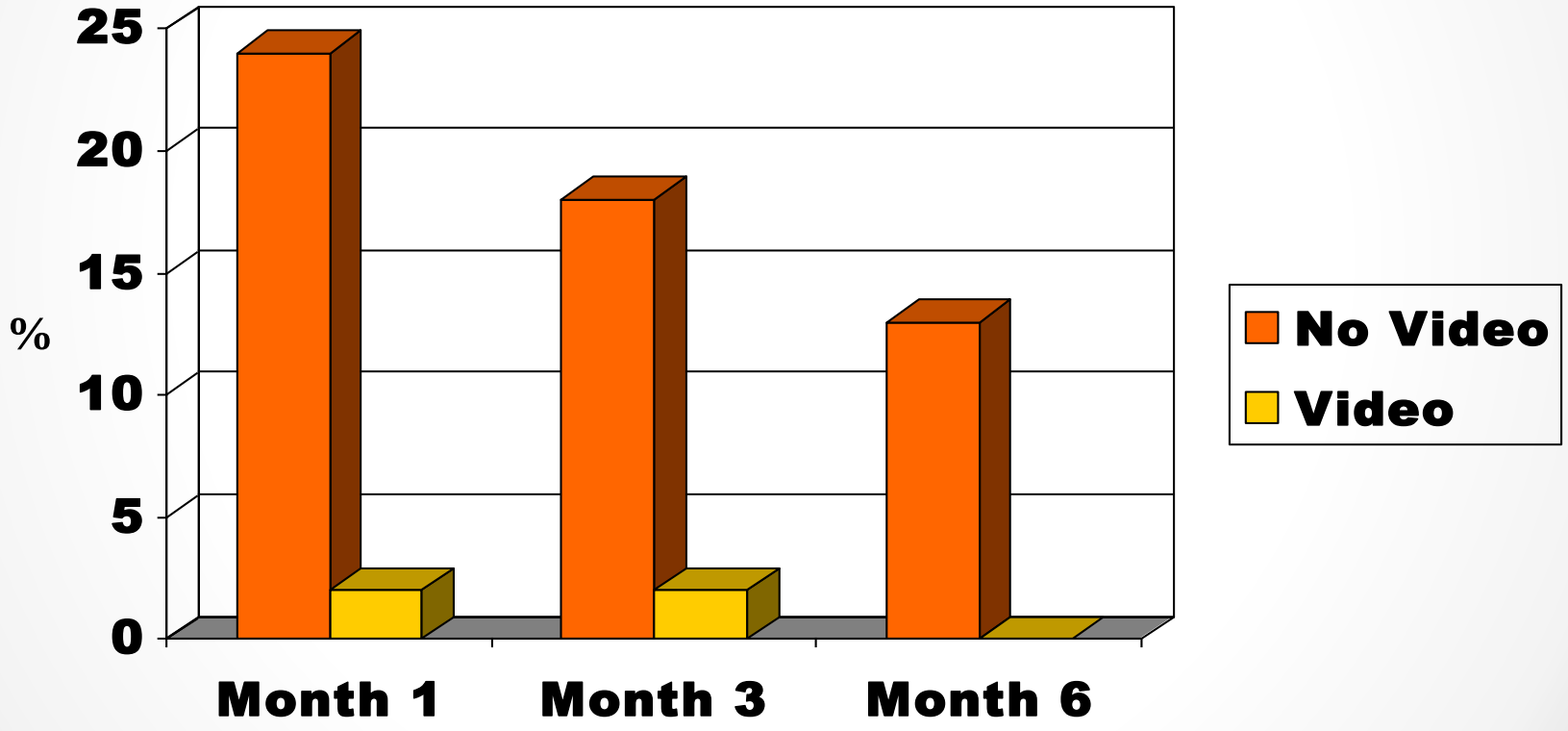
Taking Narcotics



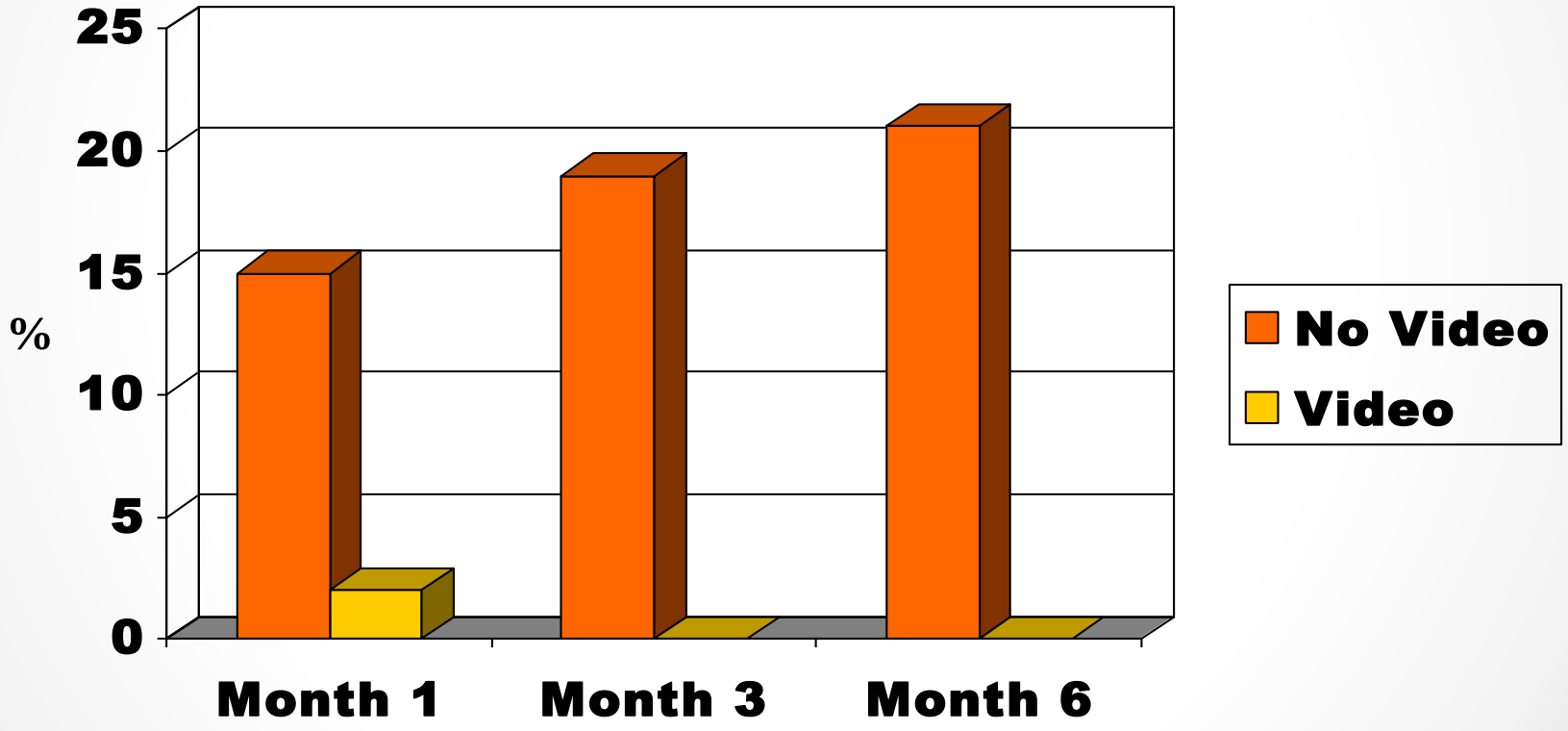
Taking Muscle Relaxant



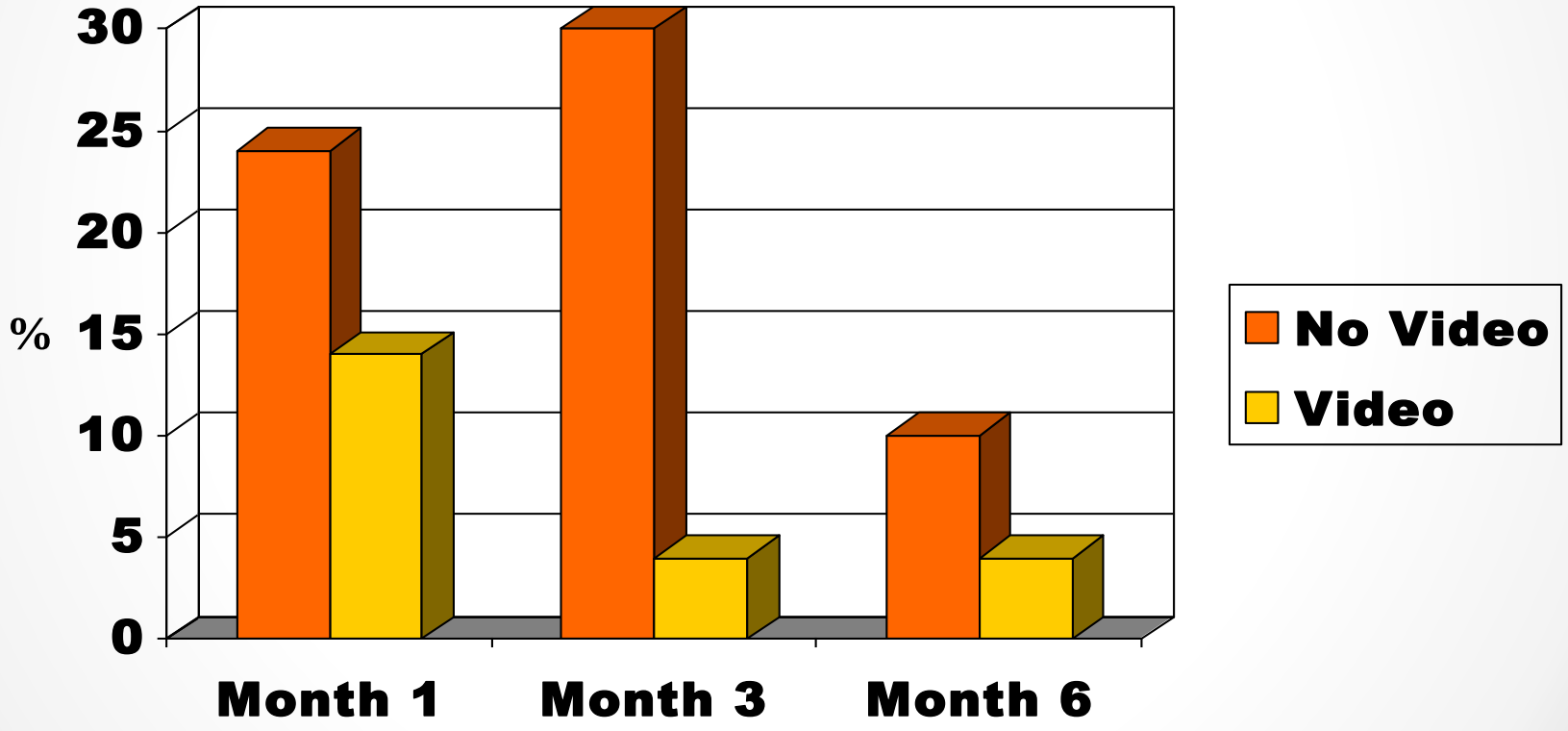
Wearing Neck Brace



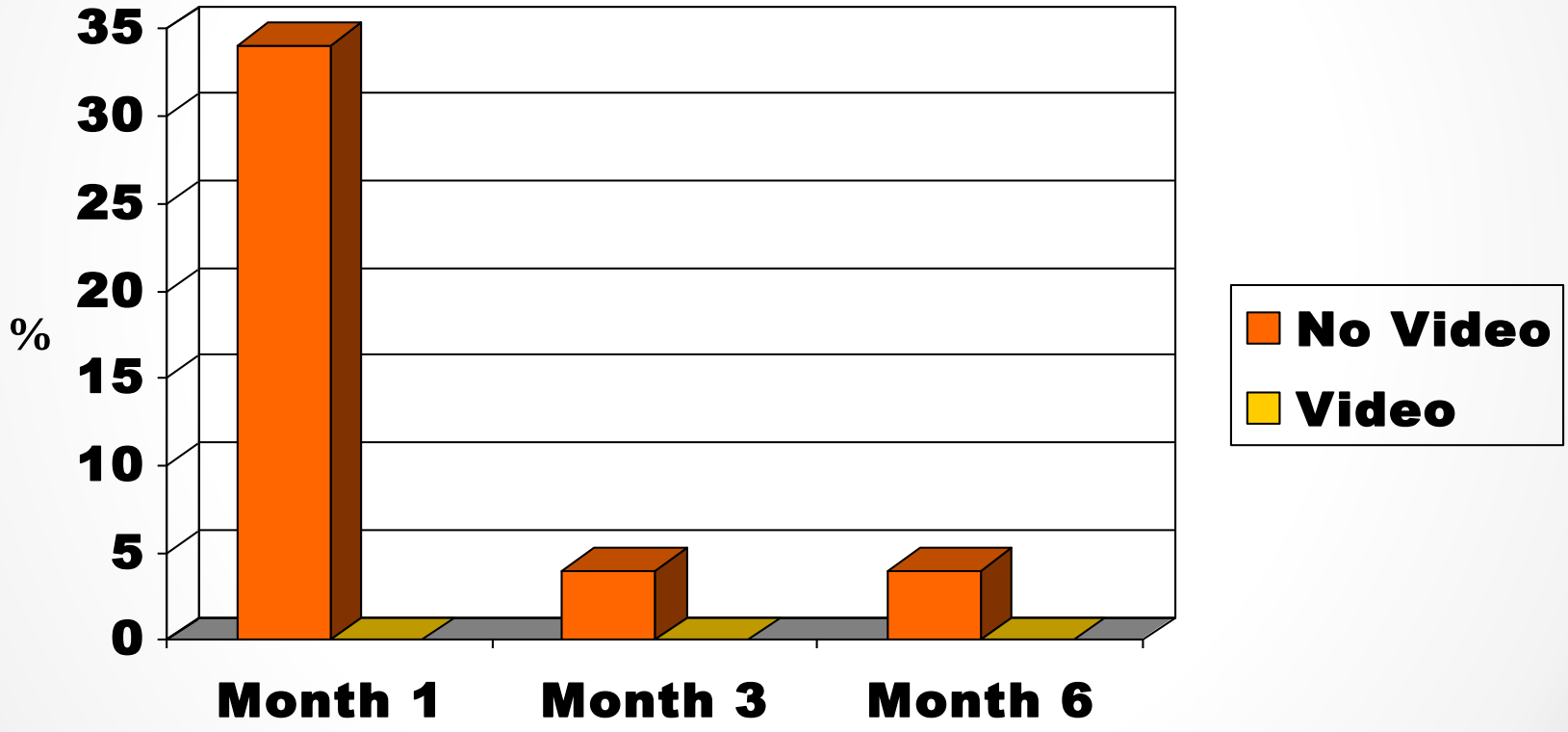
Surgical Consultation



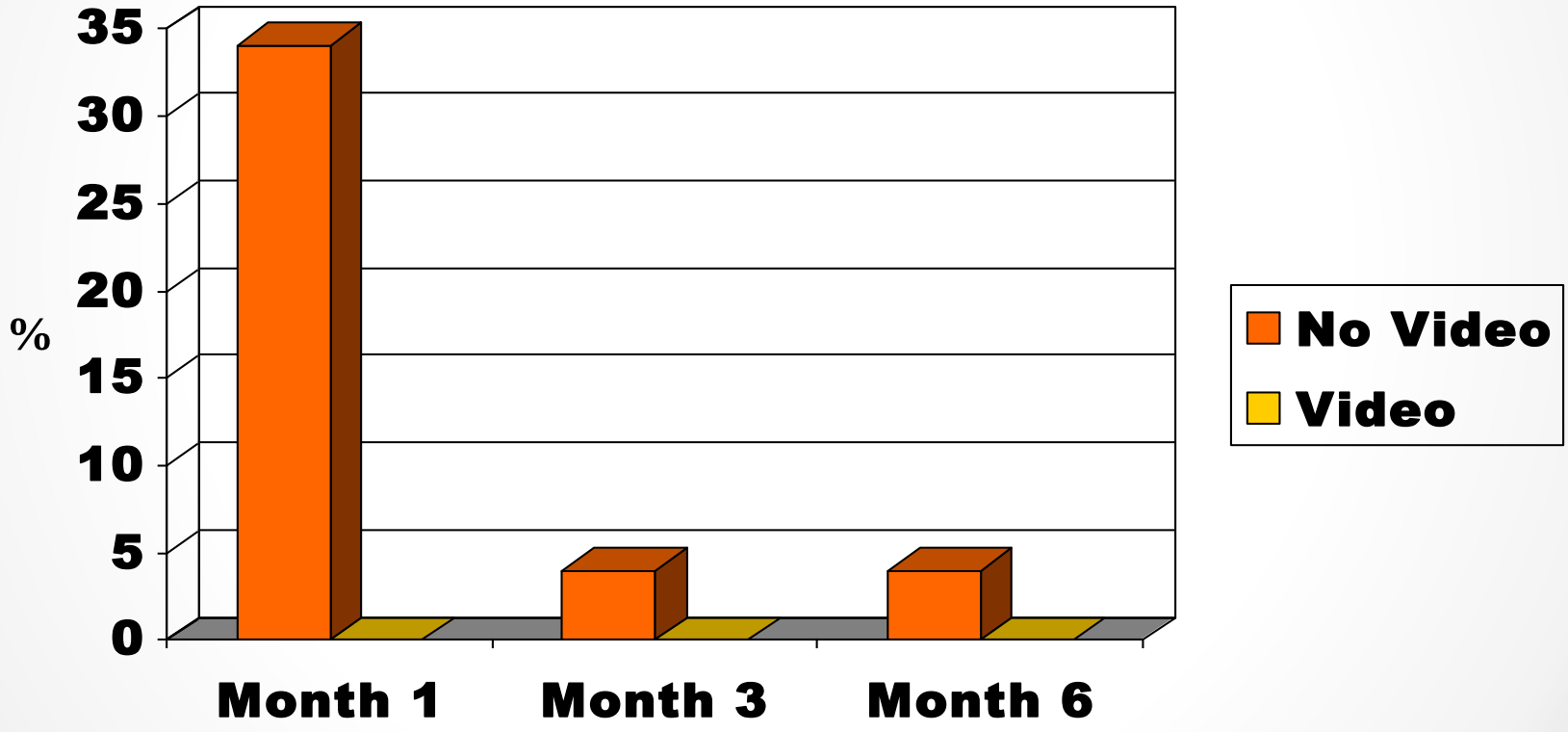
Cut Back Activities



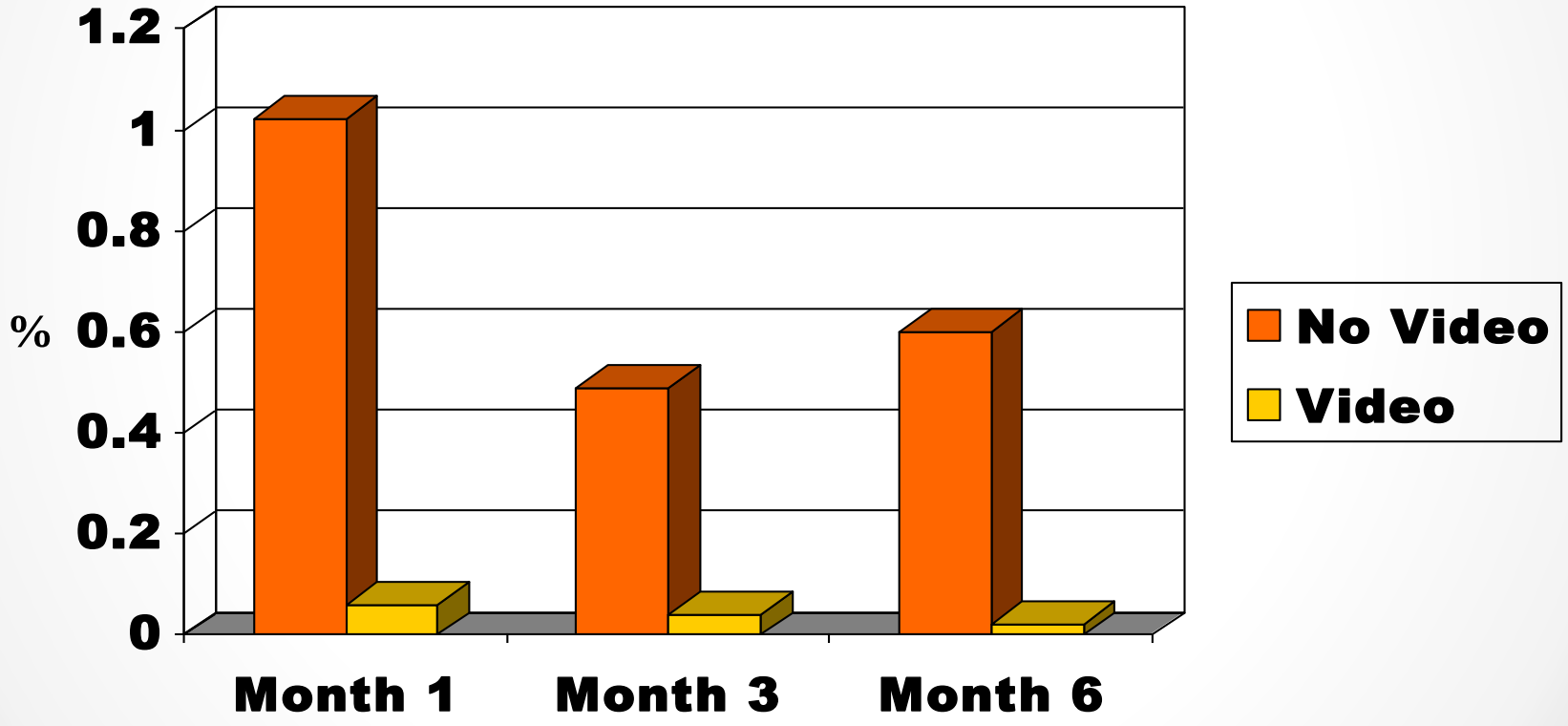
Bed Rest



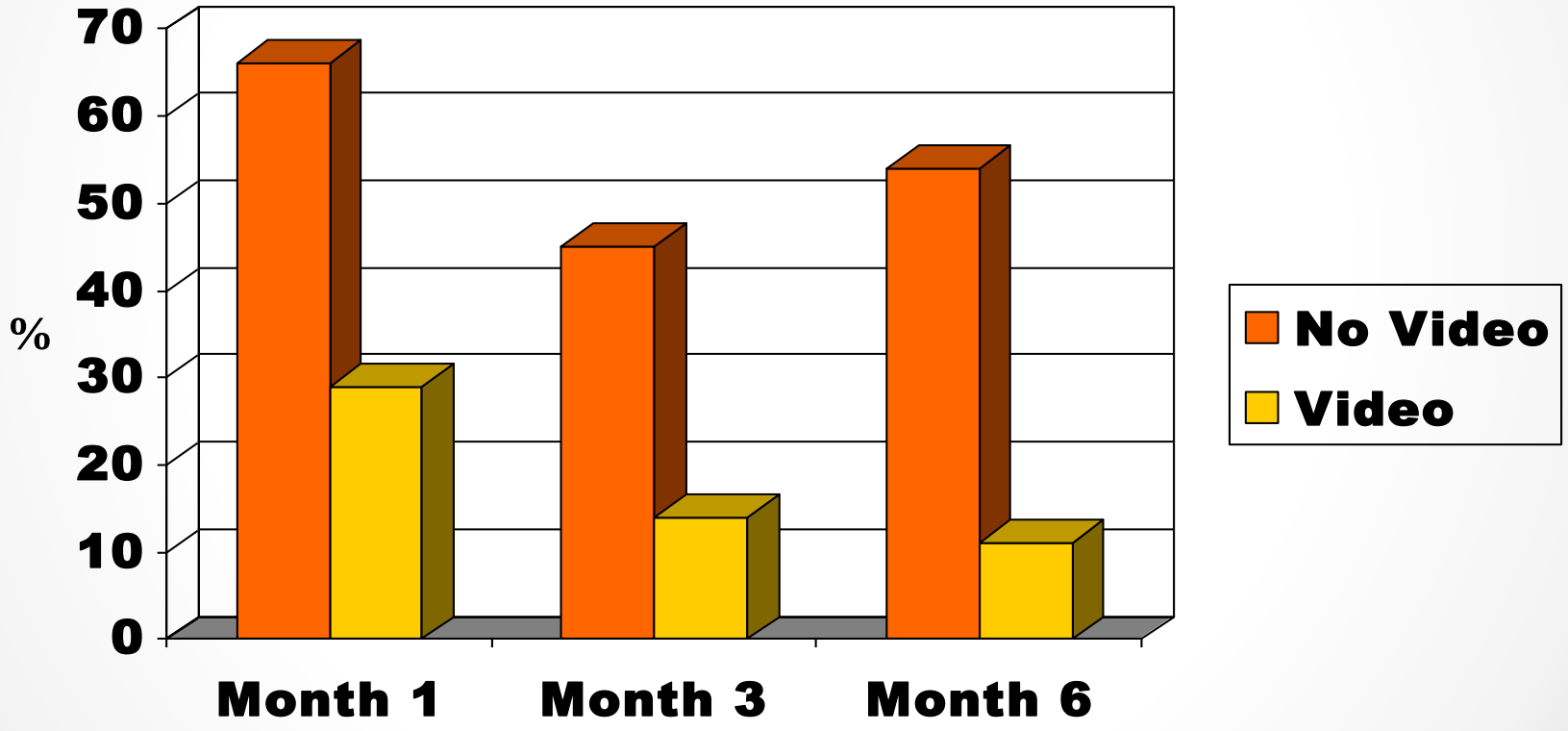
Bed Rest



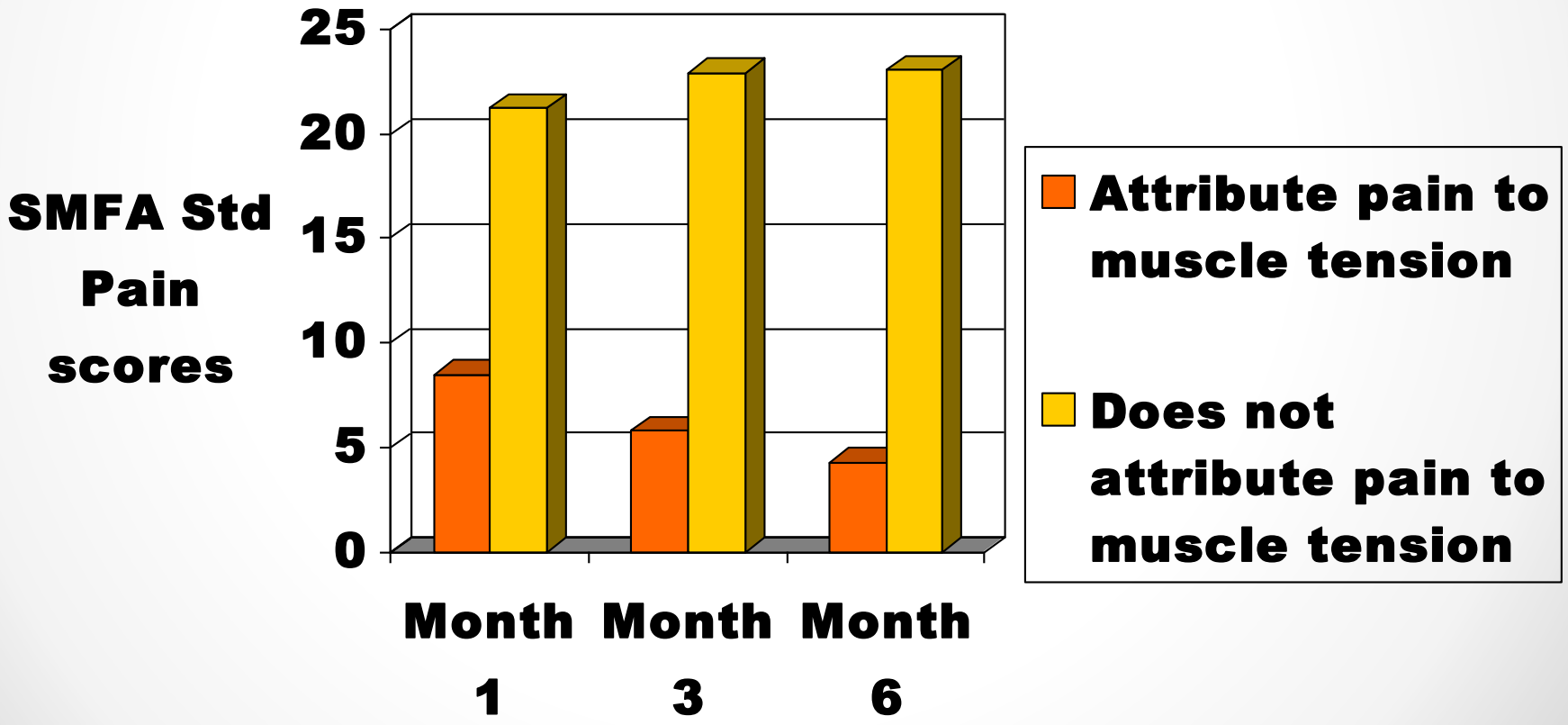
Number Missed Workdays Due to Injury



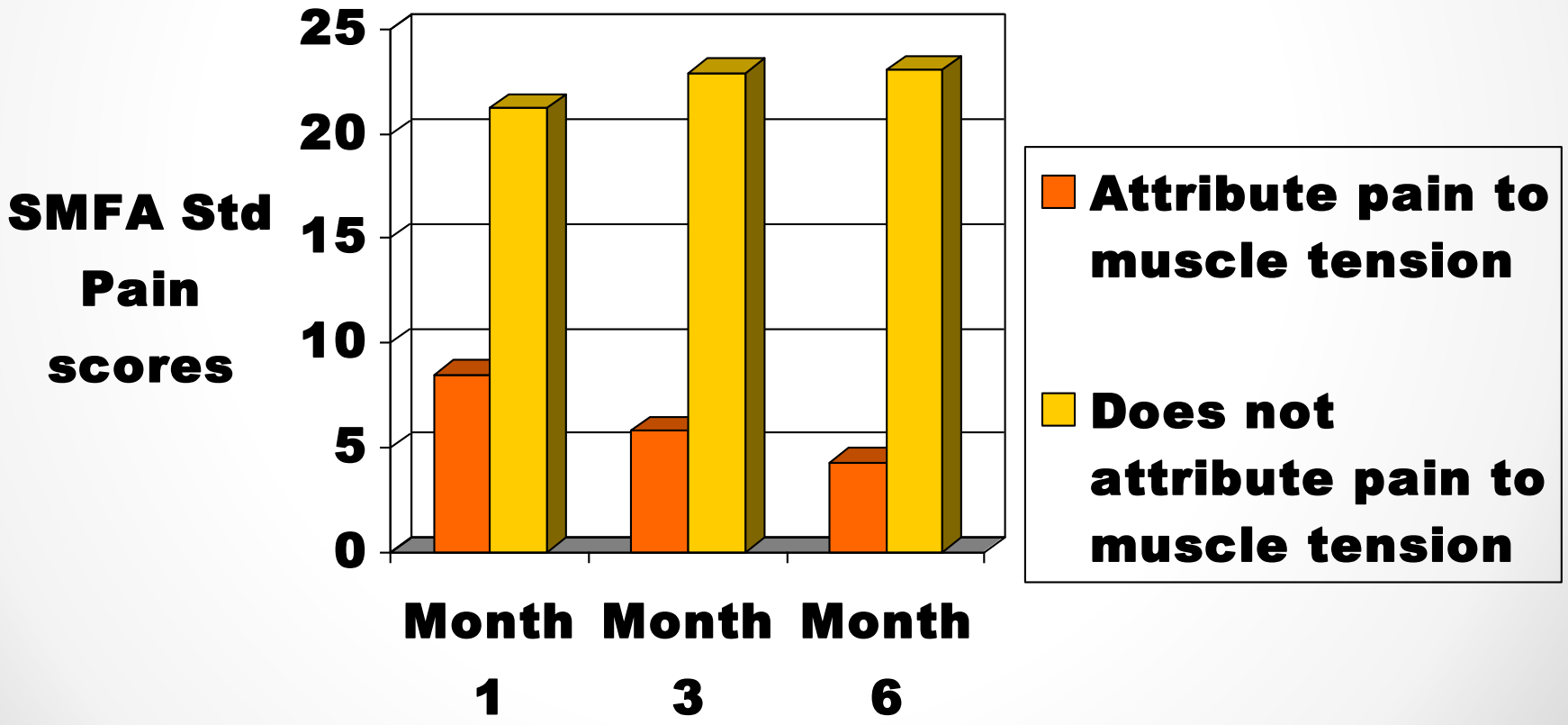
Primary Care Doctor Office Visits



attribution of pain etiology and pain improvement



attribution of pain etiology and pain improvement



Why was a 12 minute video so effective in preventing chronic neck/head pain?

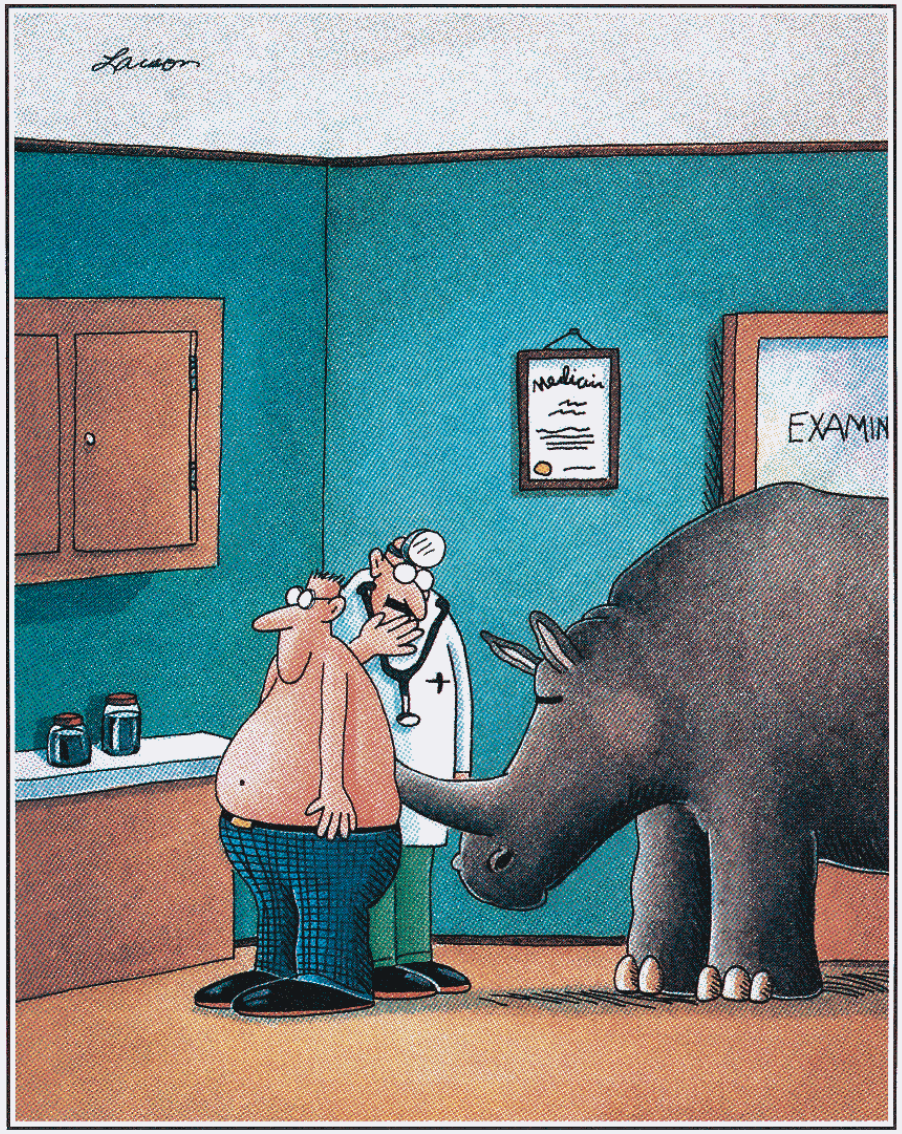
- Answer is the core of this talk

Where Does the Pain Come From?

- Key question-seldom asked
- Is there a strong central component?
  - Is the pain a neurological construction absent peripheral mechanisms?
  - Evidence of central factors
    - Seratonergic systems
    - Dopaminergic systems
    - Antidepressant medication
    - Imaging studies
    - Small % of the variance
- Muscle Fatigue
  - No evidence for byproducts which would indicate fatigue
- Peripheral Mechanisms- *Trigger Points*

Typical Diagnosis

- Cervical Strain
- Lumbar Strain
- Repetitive Strain Injury
- Tension Headache
- TMJ or TMD
- Myofascial Pain Syndrome



Wait a minute here, Mr. Crumbley, maybe its not kidney stones after all!

Typical Misdiagnoses

- Ruptured or bulged disc
- Pinched nerve
- Carpal tunnel syndrome
- Tennis elbow
- Bursitis
- Thoracic Outlet Syndrome
- Depression (or other Psychiatric diagnosis)
- Fibromyalgia

Etiological Theories

- Fatigue/Posture models
- Inflamation models
- Micro-lesion
- Subtle metabolic abmormality
- Trigger Points

Travell & Simons'

Myofascial Pain and Dysfunction:

The Trigger Point Manual

VOLUME 1. Upper Half of Body

Second Edition

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Consultant, Myofascial Pain and Dysfunction

Illustrations by Barbara D. Cummings

with contributions by Diane Abeloff and Jason Lee

Williams & Wilkins
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† Dr. Janet Travell's genius and medical insight identified in the first edition the clinical picture of individual myofascial pain syndromes and many perpetuating factors. In addition, we were most fortunate to have had the benefit of her advice in preparing some of this edition. She emphasized the importance of including a new chapter that covers the respiratory muscles and supplied unique pearls of clinical wisdom that sprinkle this revision.

Trigger Points (TrPs) I

- Trigger point is the sine qua non of Myofascial Pain Syndrome (MPS)
- Associated stiffness
- Localized point tenderness in muscle
- Stimulation produces local and referred pain
- Often with a palpable taut band

TrPs II

- Twitch
- Trigger because like a gun trigger is initiated with pressure
- Produces pain in another place-(target)

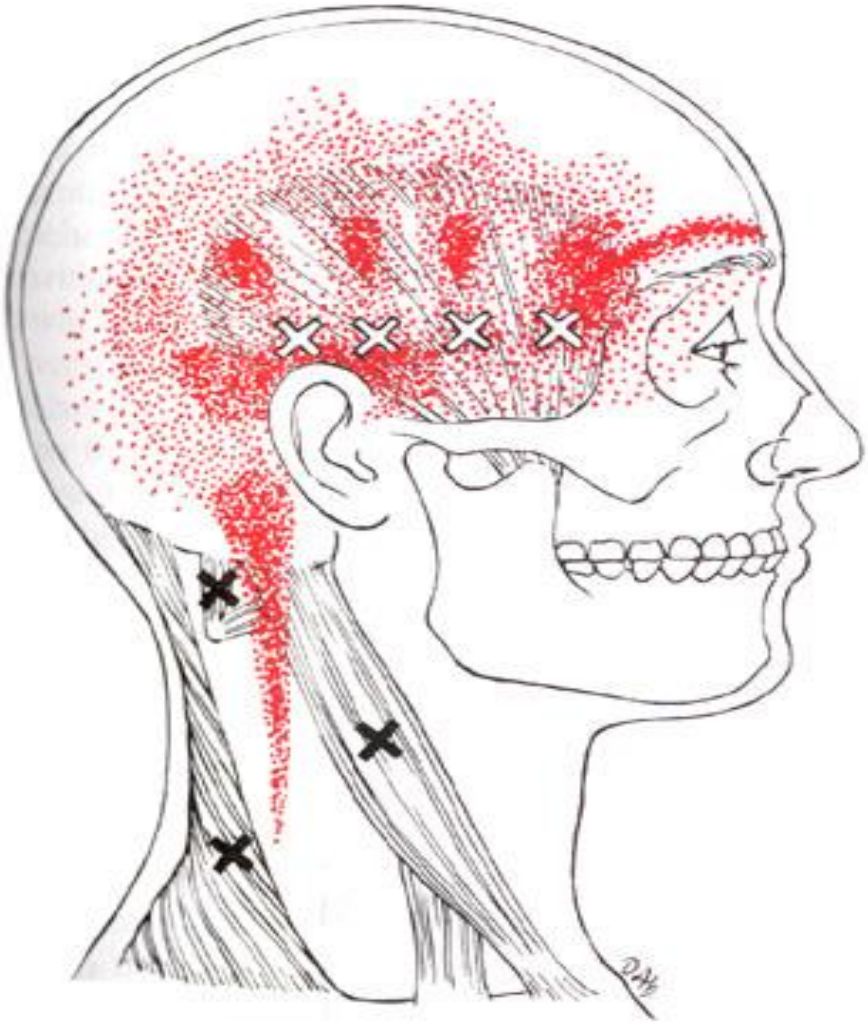
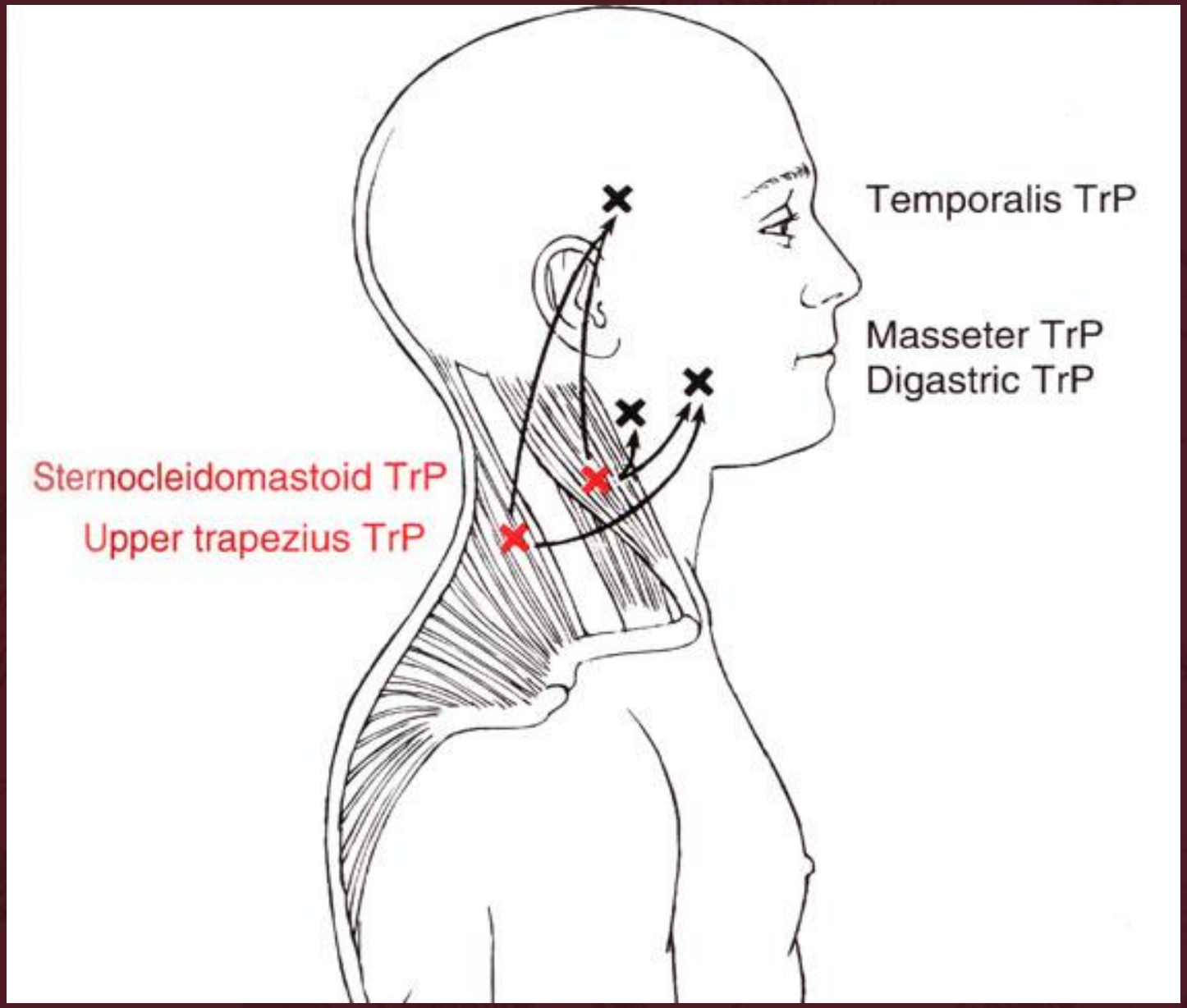


Figure 5.2. Overlapping pain referral patterns *(red)* from myofascial trigger points (**X**s) in various masticatory and cervical muscles produce typical unilateral or bilateral migraine or tension-type headache pictures.


Temporalis TrP
Masseter TrP
Digastric TrP
Sternocleidomastoid TrP
Upper trapezius TrP

toid attachment of the sternocleidomastoid. They reported inducing referred head pain by applying digital pressure to these tender muscles and by injecting hypertonic salt solution into them, location unspecified.

Sternal Division
(Fig. 7.1*A*)

An active TP in the *lower end* of the sternal division refers pain downward over the upper portion of the sternum (Fig. 7.1*A*). This is the only downward reference of pain from this muscle.[49, 53] True trigeminal facial neuralgia is not accompanied by sternal pain, which, when also present, suggests the sternocleidomastoid myofascial syndrome.

When an unusual TP is activated in the lowest part of the sternal division, where that division may merge with a slip of the inconstant sternalis muscle, the TP is associated with a paroxysmal dry cough that can be precipitated by mechanical stimulation of the TP.

At the *mid-level* of the sternal division, TPs refer pain homolaterally, arching across the cheek (often in finger-like projections) and into the maxilla, over the supraorbital ridge and deep within the orbit.[61] Pain may be referred on the same side to the external auditory canal.[56, 62] The quality of the pain is described by patients to be aching as in the deep pain defined by Kellgren.[23] The TPs along the inner margin at the mid-level of this division refer pain to the pharynx and to the back of the tongue during swallowing,[5] which causes "sore throat," and to a small round area at the tip of the chin.[53] Marbach [28] shows a similar pattern that includes the cheek, temporomandibular joint and mastoid areas.

In the *upper end* of the sternal division, TPs refer pain to the occipital ridge behind, but not close to the ear, and to the vertex of the head like a skull cap, with scalp tenderness in the pain reference zone.

Autonomic concomitants of TPs in the sternal division relate to the homolateral eye and nose.[49, 53] Eye symptoms include excessive lacrimation, reddening (vascular engorgement) of the conjunctiva, apparent "ptosis" (narrowing of the palpebral fissure) with normal pupillary size and reactions, and visual disturbances. The "ptosis" is due to spasm of the orbicularis oculi muscle, rather than to weakness of the levator palpebrae muscle. The spasm is caused by increased excitability of motor units within the reference zone of sternal division TPs. The patient may have to tilt the head backward to look up, because of inability to raise the upper eyelid. Visual disturbances include not only blurring of vision,[47, 49] but also dimming of perceived

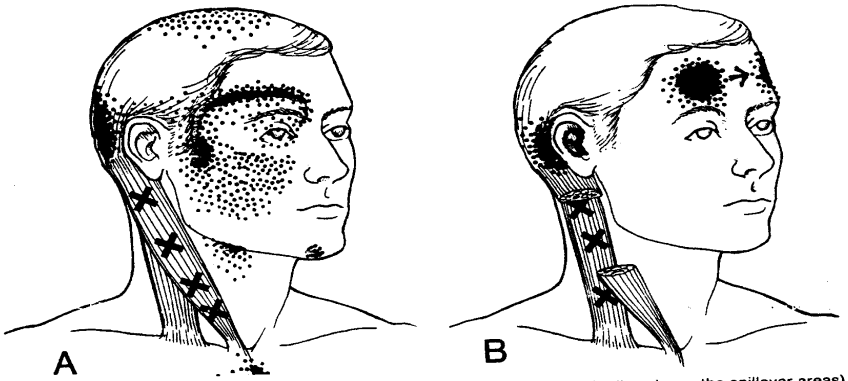


Figure 7.1. Referred pain patterns (*solid red* shows essential zones and *stippling* shows the spillover areas) with location of corresponding trigger points (Xs) in the right sternocleidomastoid muscle. *A*, the sternal (superficial) division. *B*, the clavicular (deep) division.

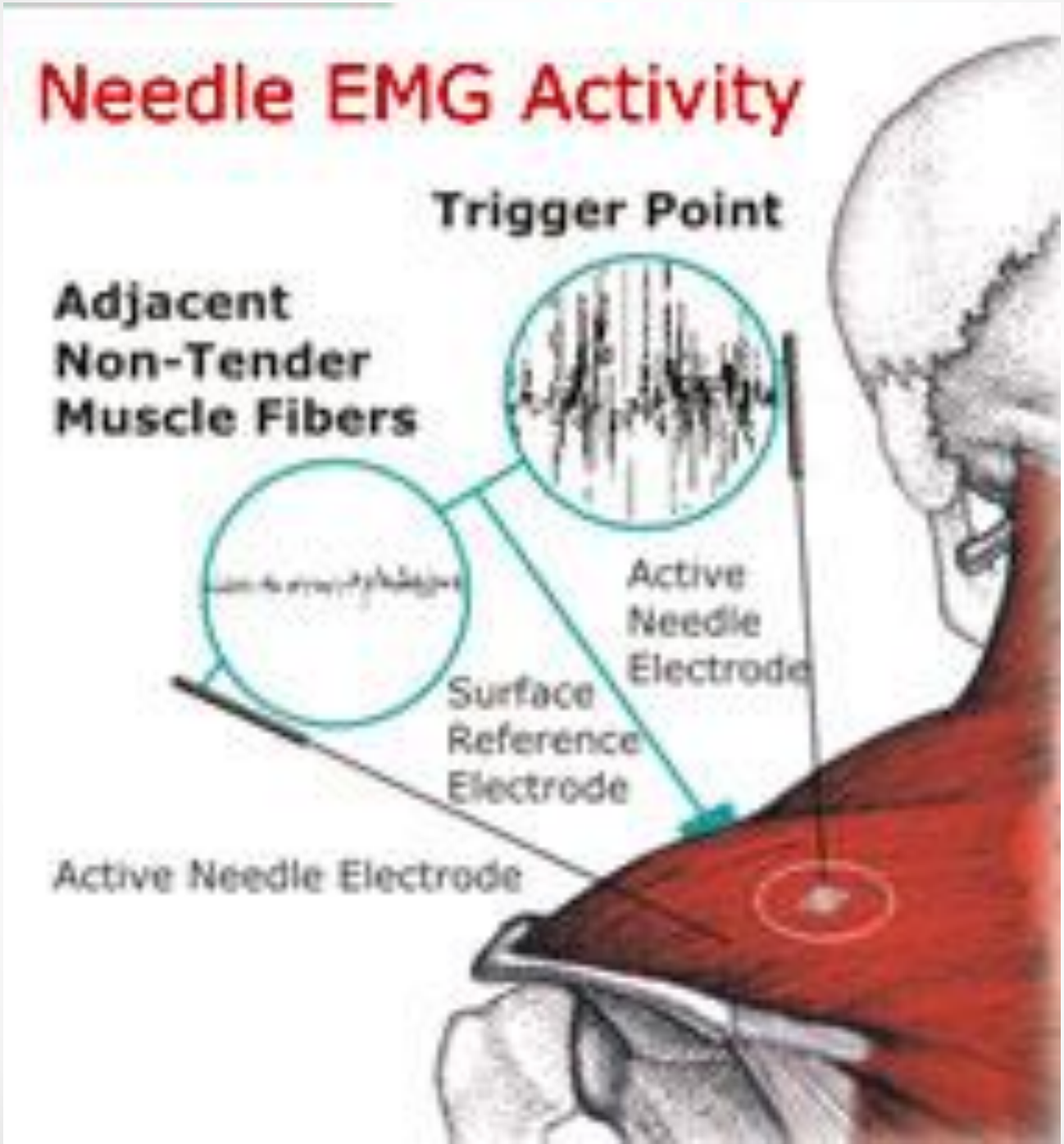
rized in Table 8-2. In 1992, Wolfe *et al.*[300] reported a study part of which involved the

tients with low back pain by two examiners picked from a pool of one general practitioner and four medical students. Each

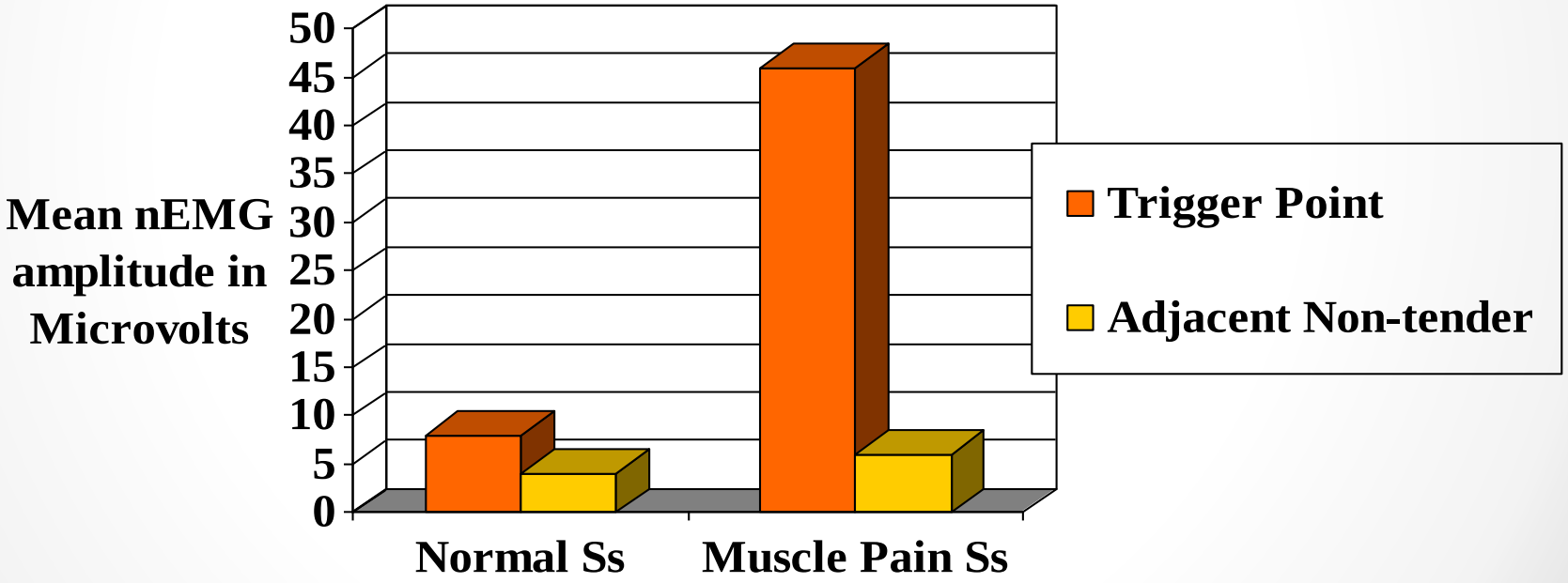
Table 8-2 ***Interrater Reliability of Examinations for Trigger Point Characteristics, Kappa Values***

Examination	*Wolfe et al.*[300]	*Nice et al.*[207]	*Njoo et al.*[210]	*Gerwin et al.*[97]	*Mean*
Spot tenderness	0.61		0.66	0.84	0.70
Jump sign			0.70		0.70
Pain recognition	0.30		0.58	0.88	0.59
Palpable band	0.29		0.49	0.85	0.54
Referred pain	0.40	0.38	0.41	0.69	0.47
Twitch response	0.16		0.09	0.44	0.23
Mean	0.35	0.38	0.49	0.74	

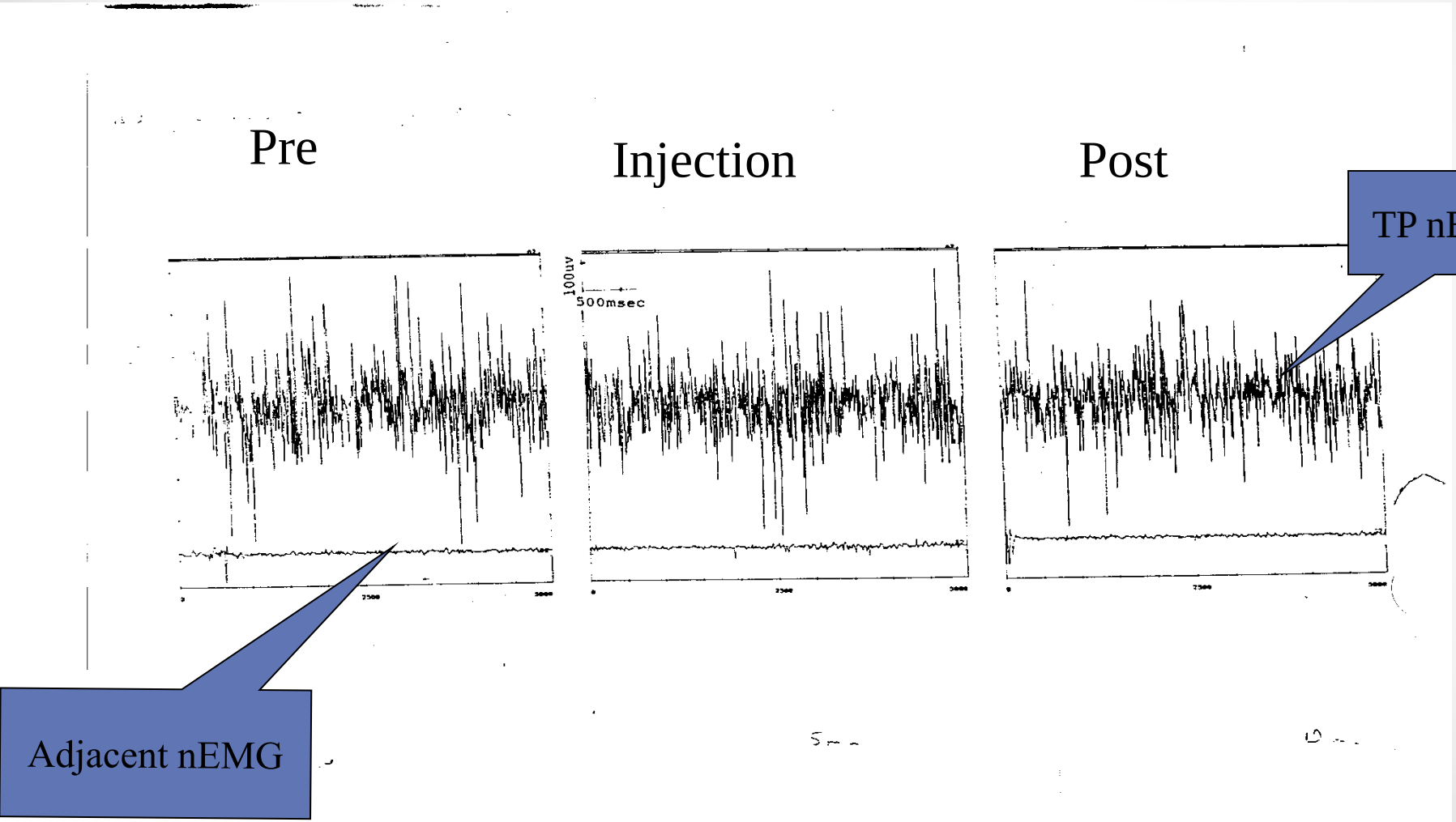
Gerwin, R., Shannon, S., Hong, C., Hubbard, D. & Gevirtz, R. (1997) Interrater reliability in myofascial trigger point examination. *Pain, 69*, 65-73.


Needle EMG Activity
Trigger Point
Adjacent
Non-Tender
Muscle Fibers
Active
Needle
Electrode
Surface
Reference
Electrode
Active Needle Electrode

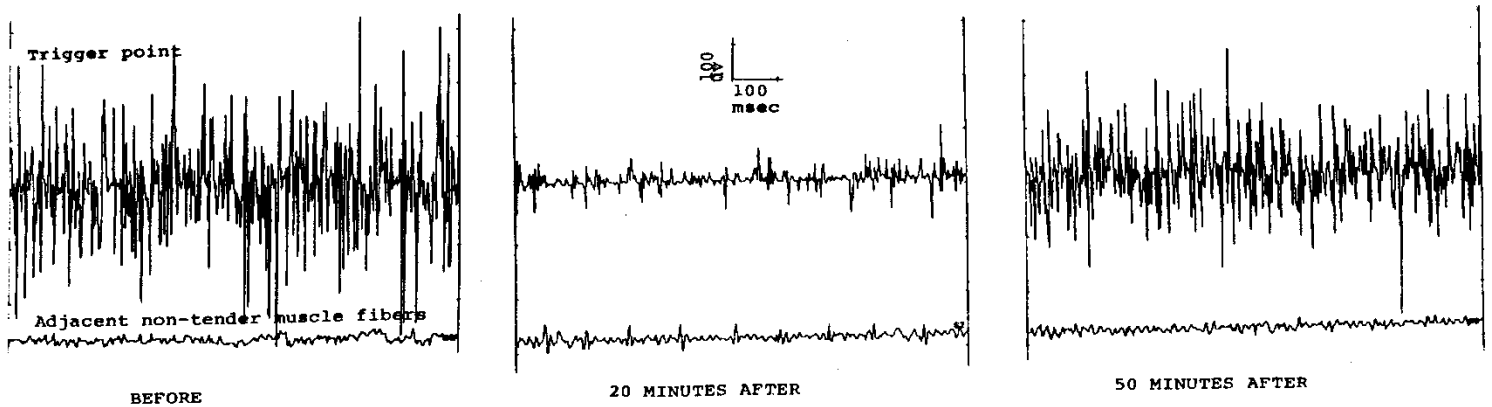
Myofascial Trigger Points Show Spontaneous Needle EMG Activity (Hubbard & Berkoff, 1993)



Effects of Curare on nEMG in TPs and Adjacent, (Non-tender) Sites



The Effect of Phentolamine Injection on TP & Adjacent nEMG



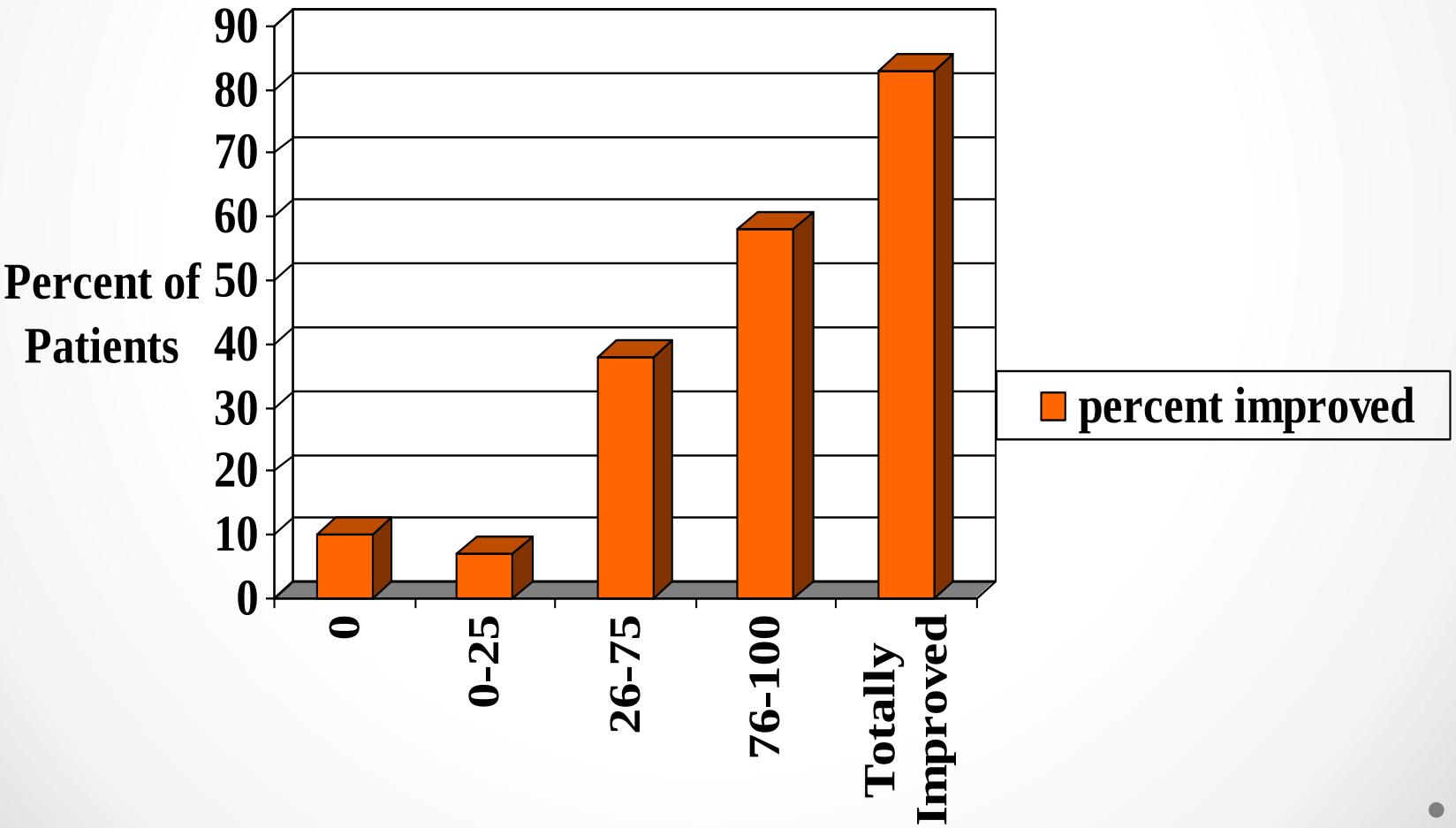
PHENTOLAMINE 2.5MG INJECTED DIRECTLY INTO TRIGGER POINT IN PATIENT WITH MYOFASCIAL PAIN
David Hubbard, MD, Dpt Neurology, University of California, San Diego

Phentolamine effect on the spontaneous electrical activity of active loci in a myofascial trigger spot of rabbit skeletal muscle*1 .

Archives of Physical Medicine and Rehabilitation , Volume 79 , Issue 7 , Pages 790 - 794

J . Chen , S . Chen , T . Kuan , K . Chung , C . Hong

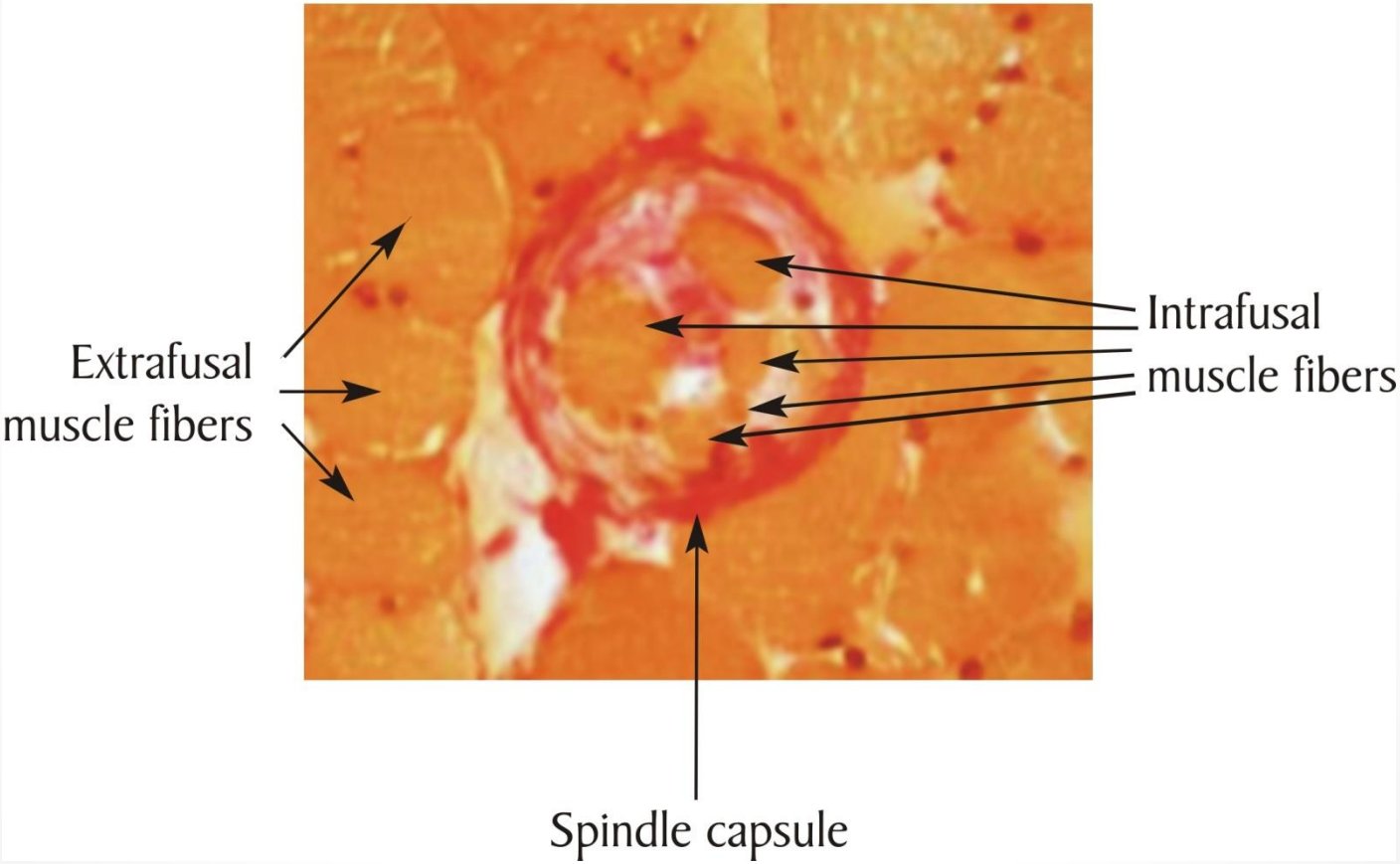
Clinical Efficacy of Phenoxybenzamine (Myotech)



Italian Spindle Studies

- Passatore, Deriu, Grassi, & Raotta (1996), *J.Auton N.S.*
- Grassi, Deriu, & Passatore (1993) *J. Physiology*
- Grassi, Deriu, Artusio, & Passatore (1993) *Arch Ital Biol*
- Grassi & Passatore(1990) *Functional Neurology*
  - Found strong response in spindle could be elicited by sympathetic cervical nerve stimulation, abolished by alpha-adrenergic blockade, unaffected by sympathetically induced vasomotor changes.
  - "These data suggest that, when the sympathetic nervous system is activated under physiological conditions, there is a marked depression of the stretch reflex which is independent of vasomotor changes and is probably due to decrease in sensitivity of muscle spindle afferents" (Grassi, Deriu, & Passatore, 1993, p.163)

Needle EMG-Guided Biopsy of a Trigger Point



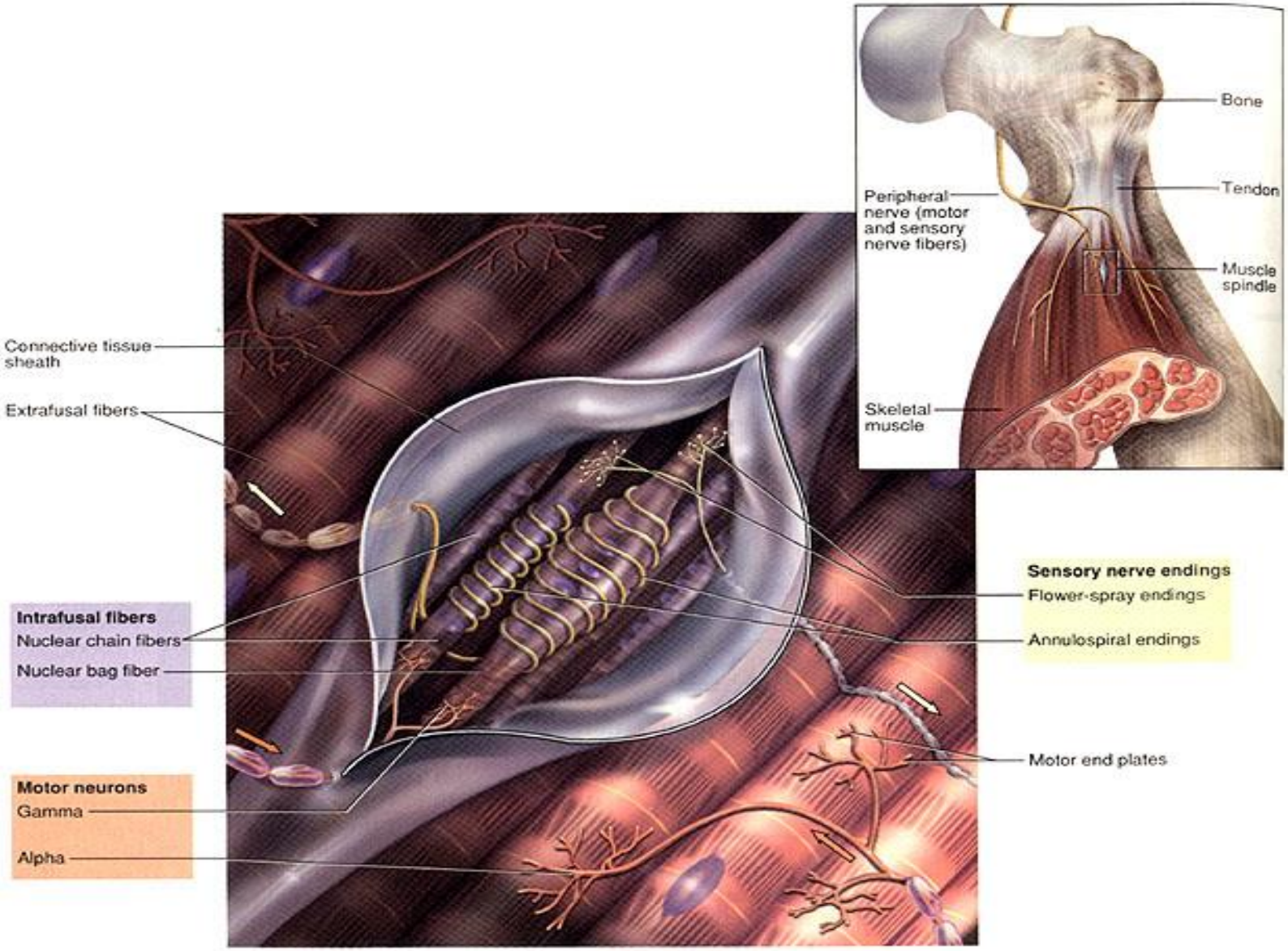


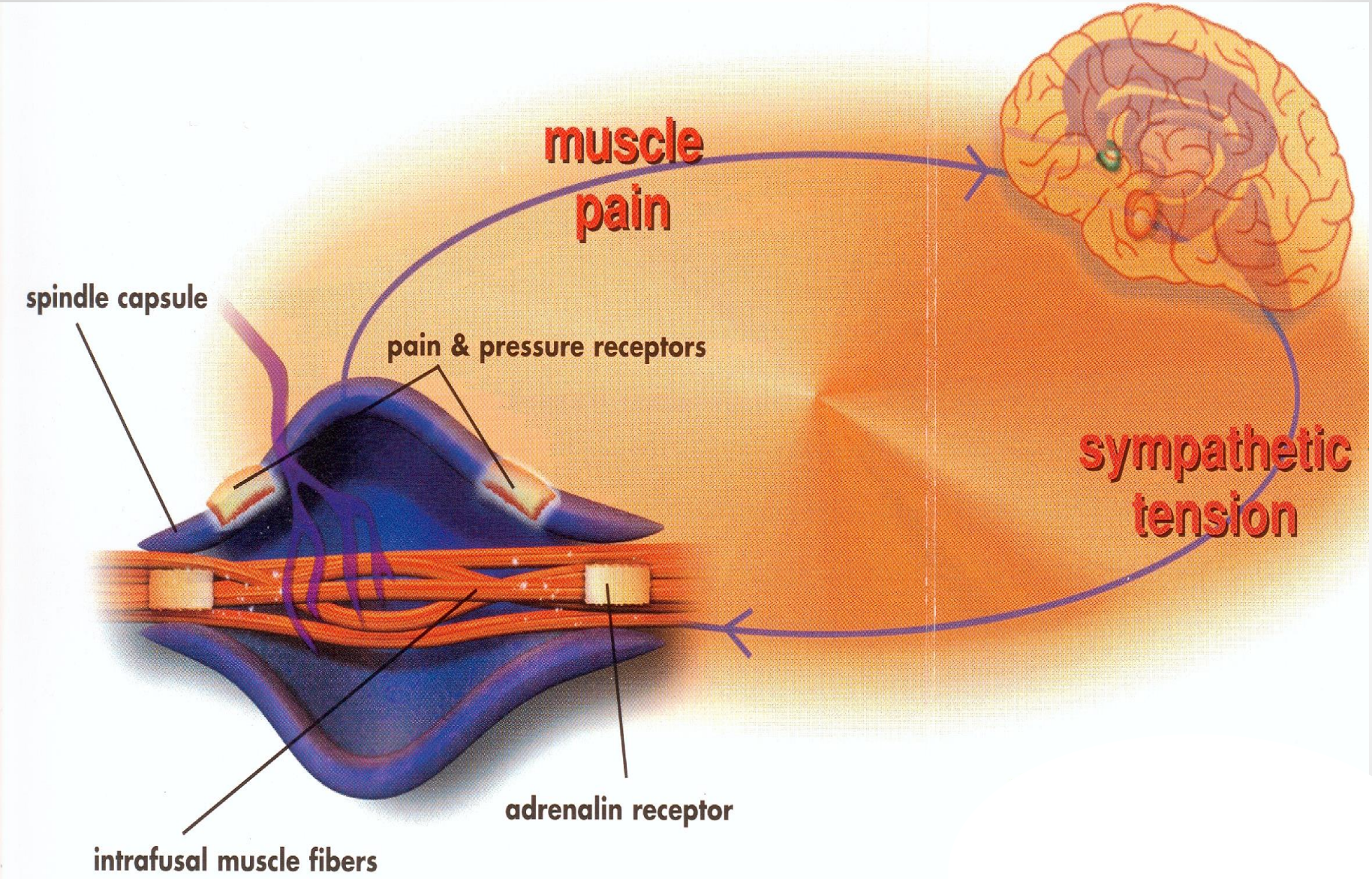
Figure 15.11 A muscle spindle and its innervation.

Muscle Spindle

Partanen, J., *J. of Physiology*, 1999. *The Fusimotor Theory Revisited*.

- "…we have observed alpha-EPS (*end plate spikes*) coactivation and even independent EPS activation, not connected to muscle contraction."

Spindles are sympathetically preparing for motor action

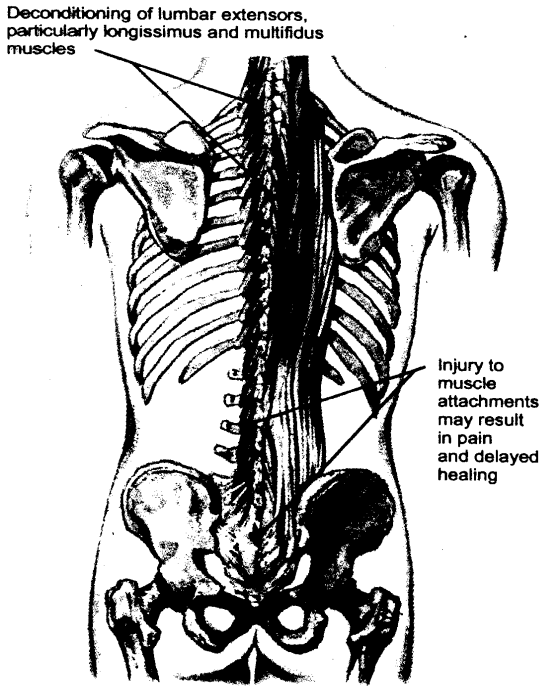

muscle
pain
spindle capsule
pain & pressure receptors
sympathetic
tension
adrenalin receptor
intrafusal muscle fibers

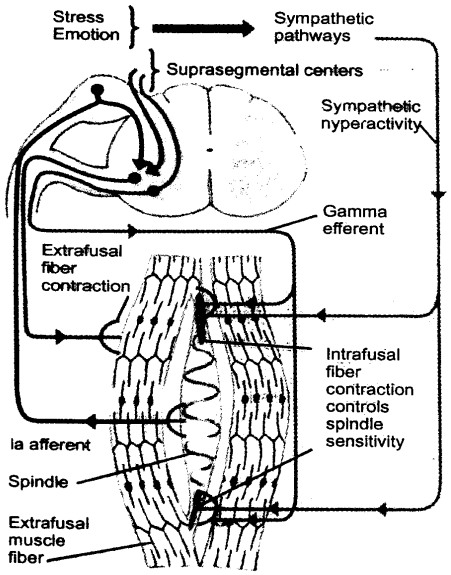
CLINICAL SYMPOSIA
ciba
Volume 48
Number 4
1996

Evaluation and Treatment of Low Back Pain

Vert Mooney, MD, Jeffery A Saal, MD, Joel S. Saal, MD

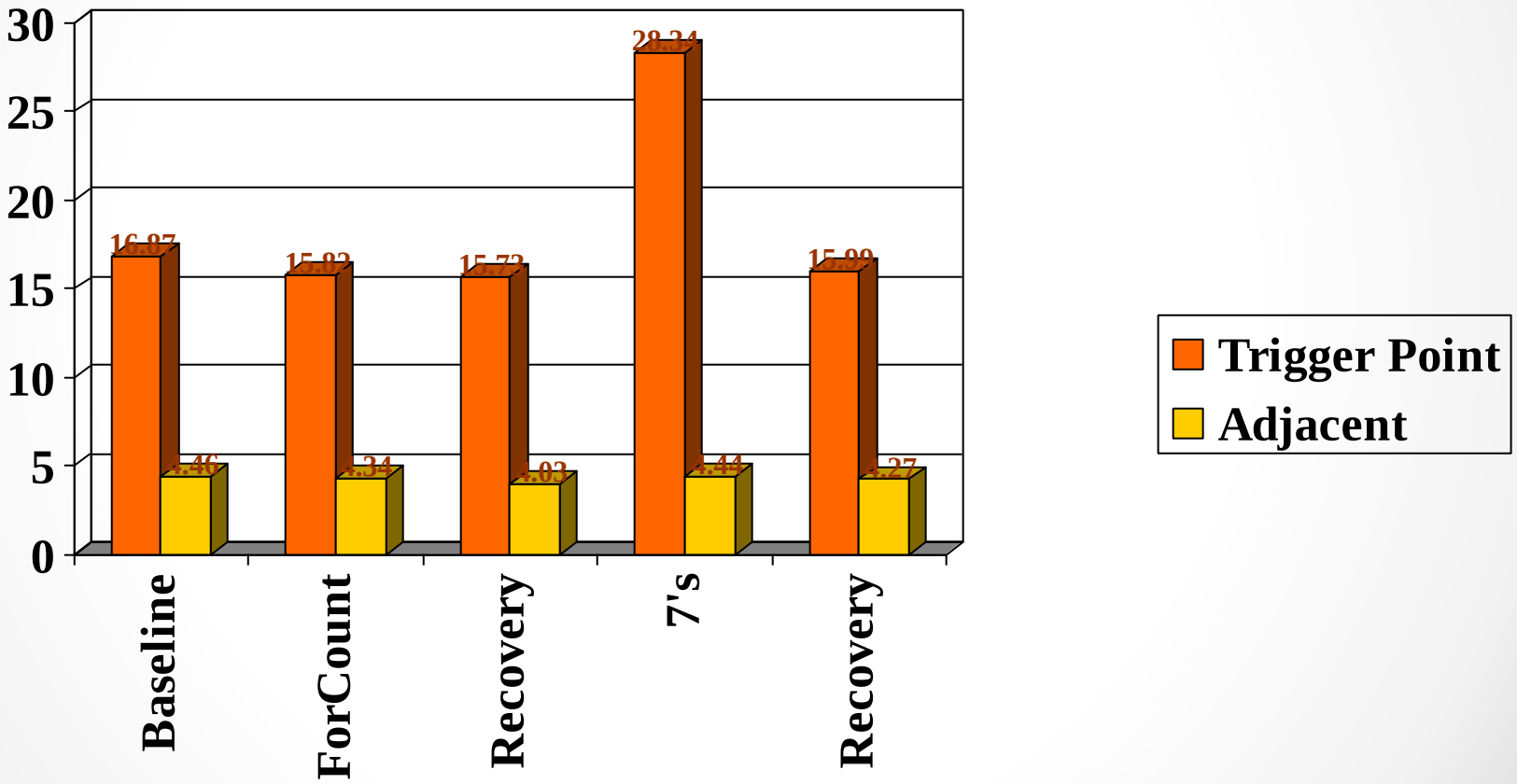
Myofascial Factors in Low Back Pain





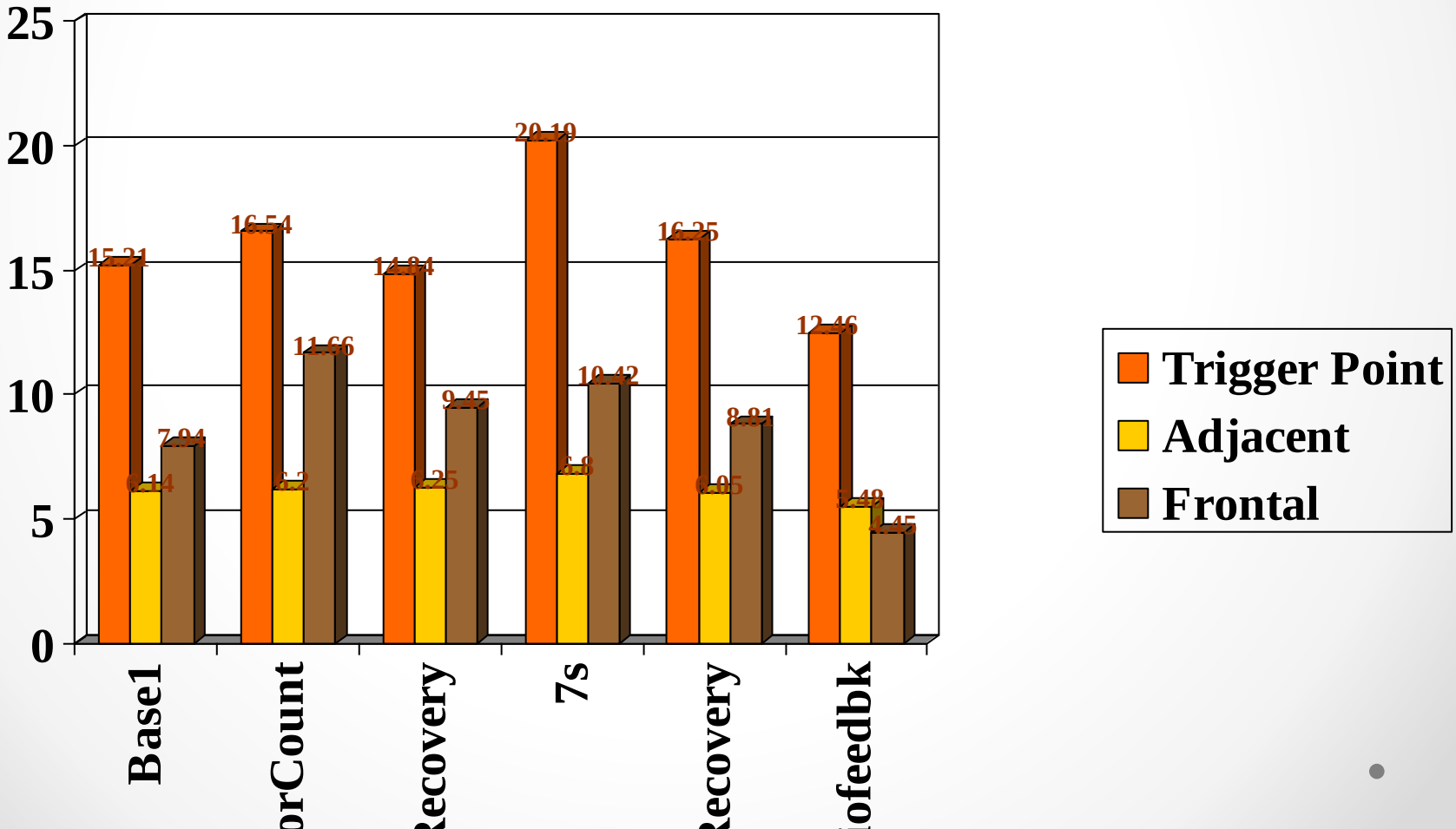
Muscle spindles provide feedback mechanism for muscle tension. Sensitivity of spindles modulated by gamma efferent system and by sympathetic innervation of spindles. Sympathetic hyperactivity can result in painful spasm of spindles.

MyoPoint

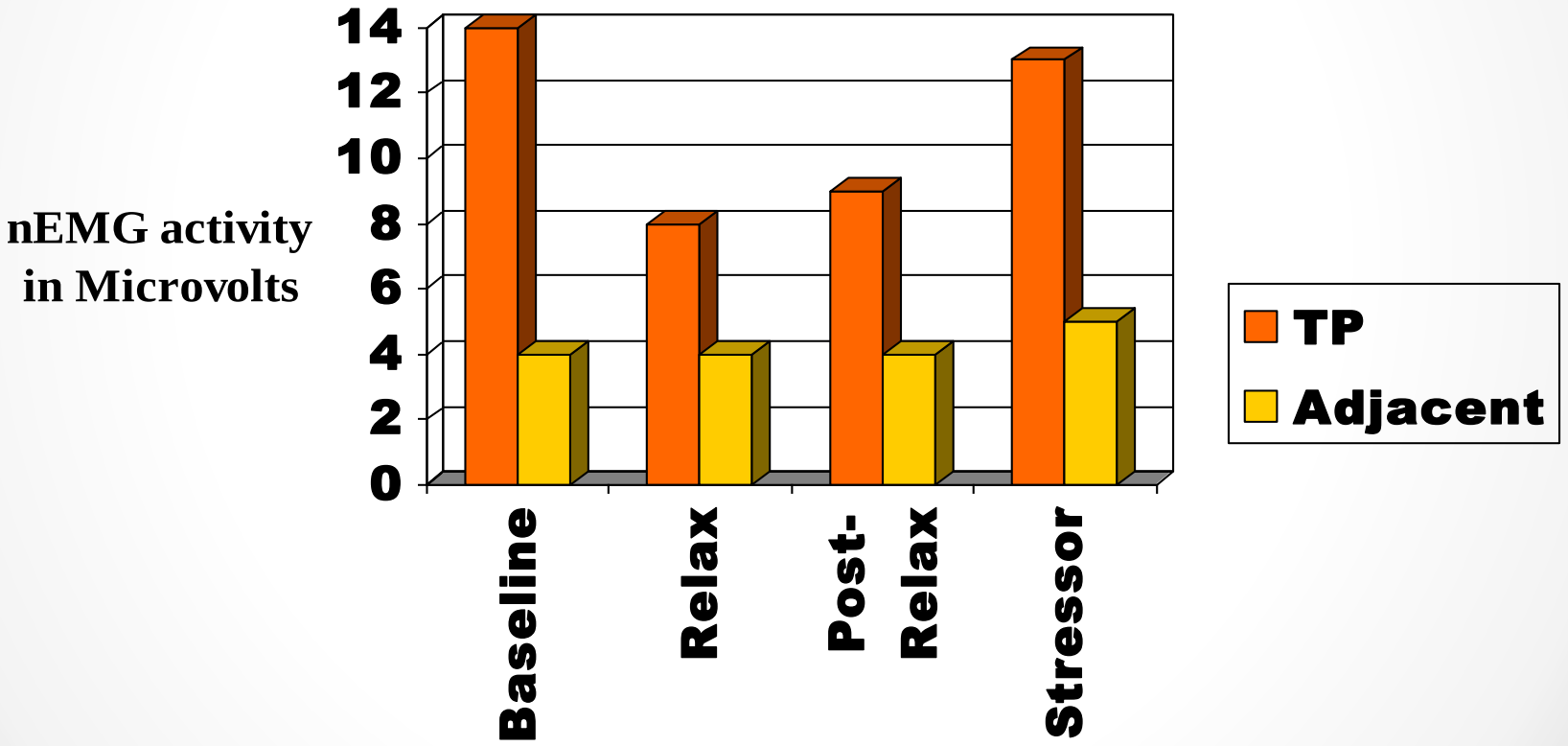

30
25
20
15
10
5
0
16.87
4.46
15.82
4.34
15.72
4.03
28.34
4.44
15.99
4.27
Baseline
ForCount
Recovery
7's
Recovery
Trigger Point
Adjacent

Trigger Point vs. Adjacent nEMG vs. Frontal EMG

Lewis, C. & Gevirtz, R.N, (1994) Needle Trigger Point and Surface EMG Measurements of Psychophysiological Responses in Tension-Type Headache Patients, *Biofeedback and Self-Regulation*, 19, 274-275 (abstract)

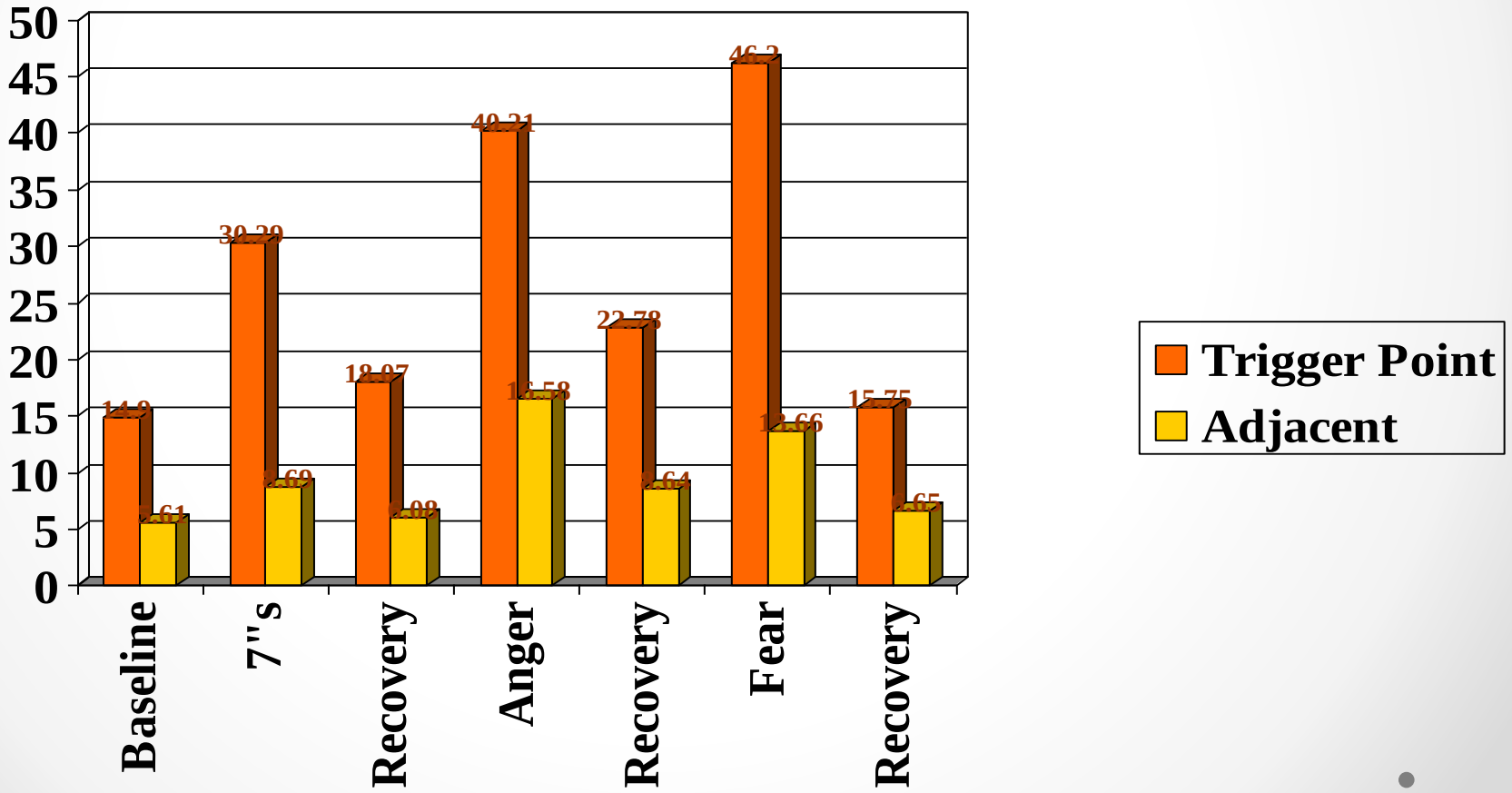


autogenic relaxation training on EMG activity in myofascial trigger points. *Journal of Musculoskeletal Pain*, 6, #4.



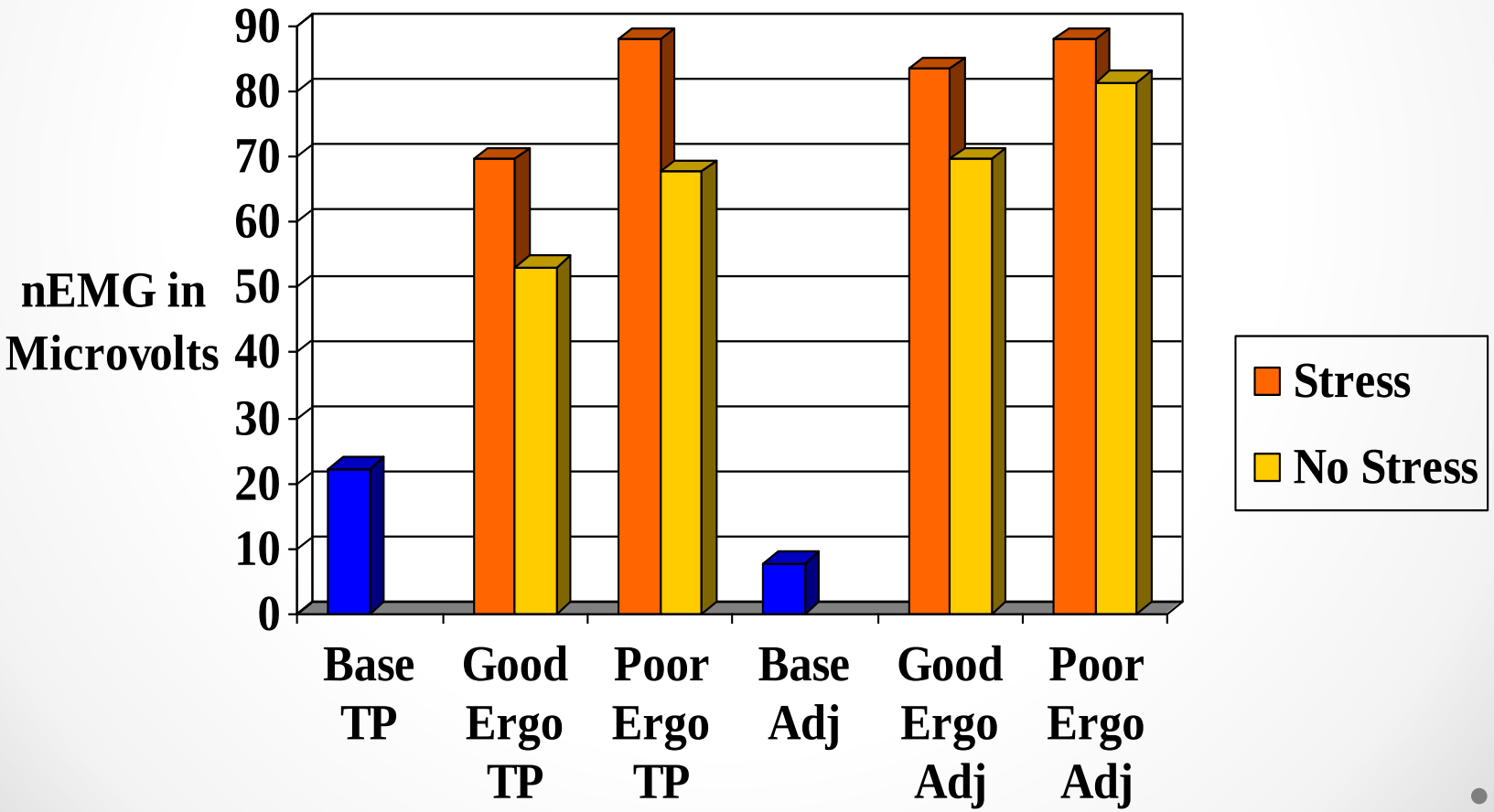
Video

Conditions Gadler, R. & Gevirtz, R.(1997) Evaluation of Needle Electromyographic Response to Emotional Stimuli *Applied Psychophysiology and Biofeedback, 22*, 137 (abstract)

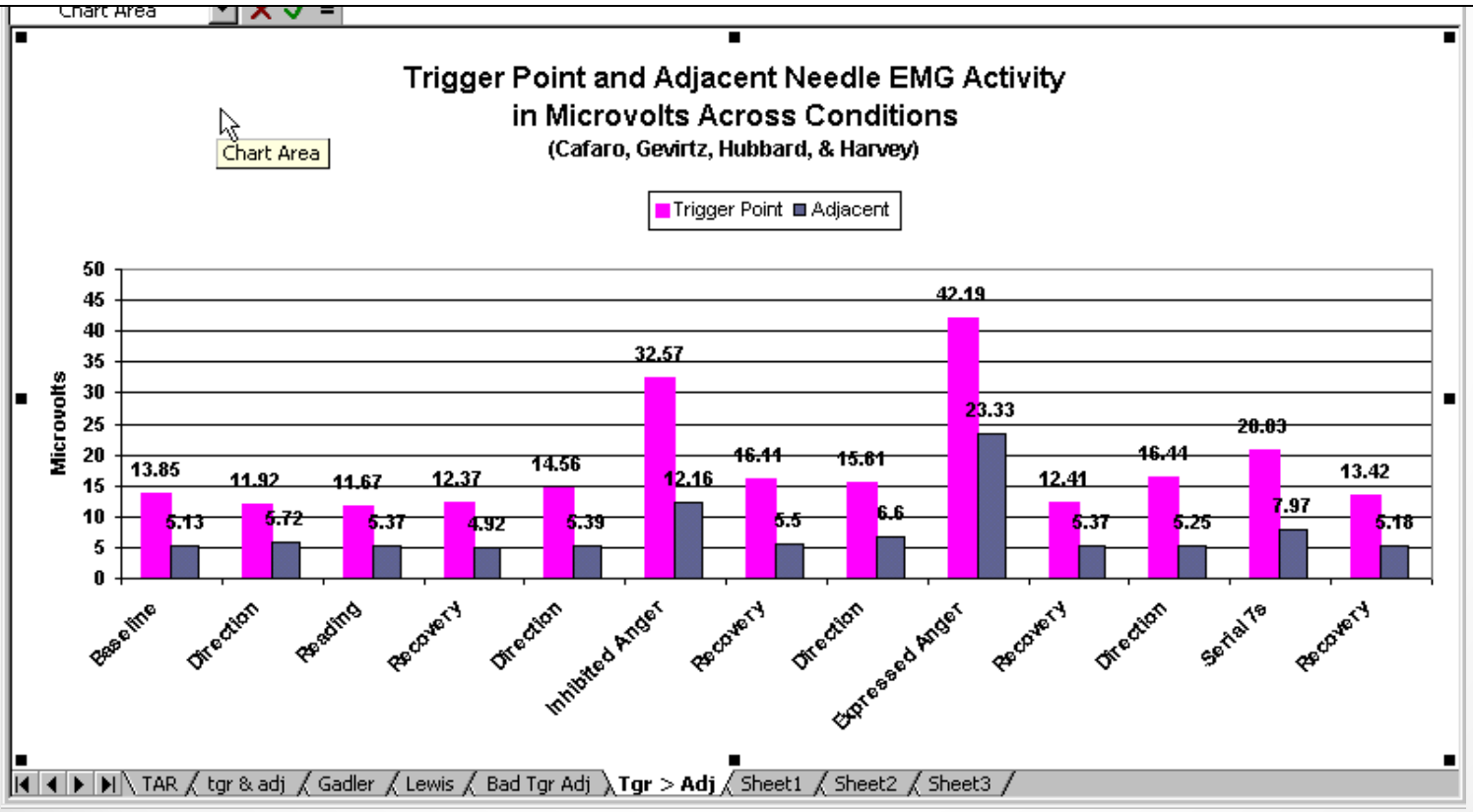


nEMG Across Ergonomic & Stress Conditions

Muse, J. & Gevirtz, R. (1999) The effects of a psychological stressor on nEMG activity while performing a typing task in good and poor ergonomic positions. *Applied Psychophysiology* and *Biofeedback, 24 (2)*, 120 (abstract).



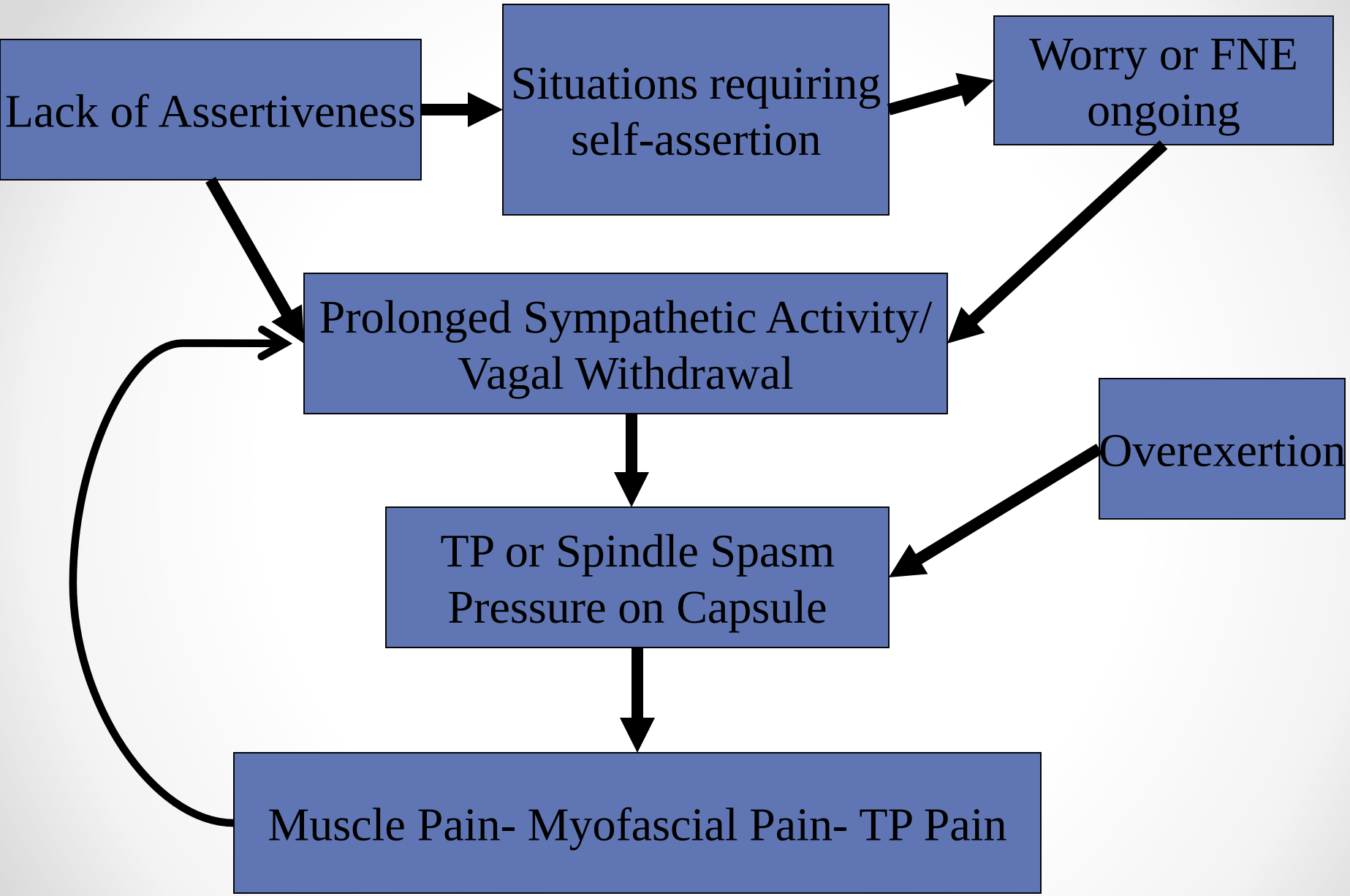
Cafaro, T.A., Gevirtz, R.N., Hubbard, D., & Harvey, M. (2001)
The exploration of trigger point and heart rate variability excitation and recovery patterns in actors performing anger inhibition and anger expression.
Applied Psychophysiology and Biofeedback, 26, 236(abstract).



Personality Traits and Tp Worsening

- In 86 1st year graduate students, the Penn State Worry Questionnaire (among a number of other predictors)predicted trigger point worsening 2-3 months later over the course of increasing stress in an academic semester, $r=.35$, $r^2=.123$ (Armm, Gevirtz, Hubbard, & Harpin, 1999)

Mediational Model of Muscle Pain



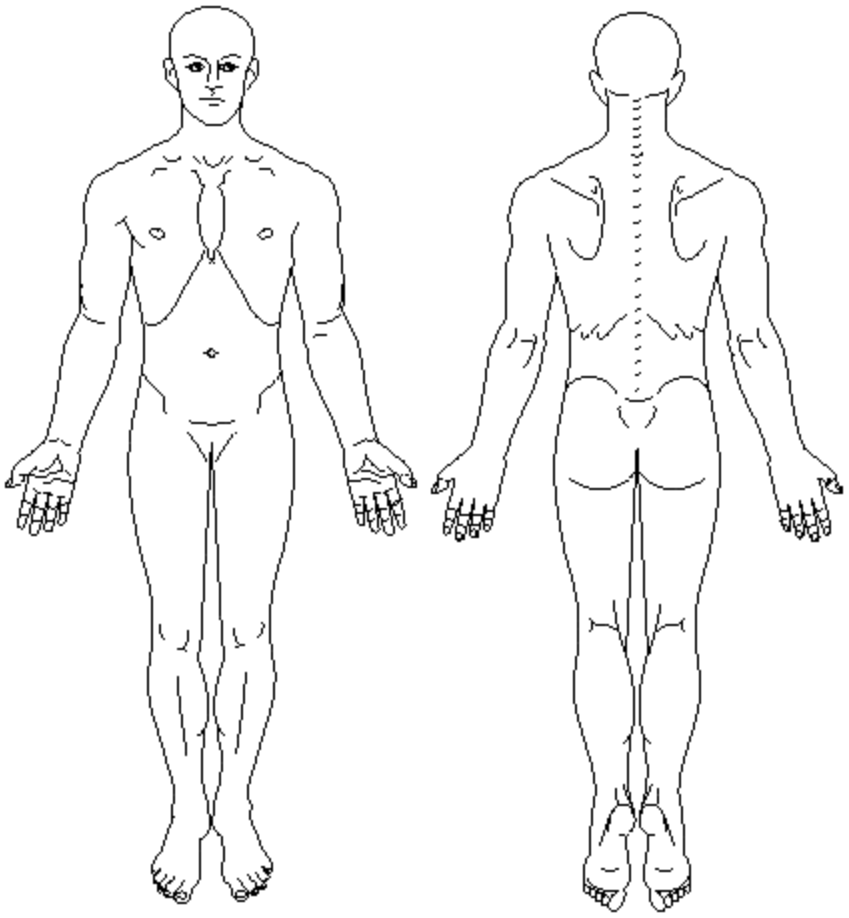
Insert Myopoint Video Here

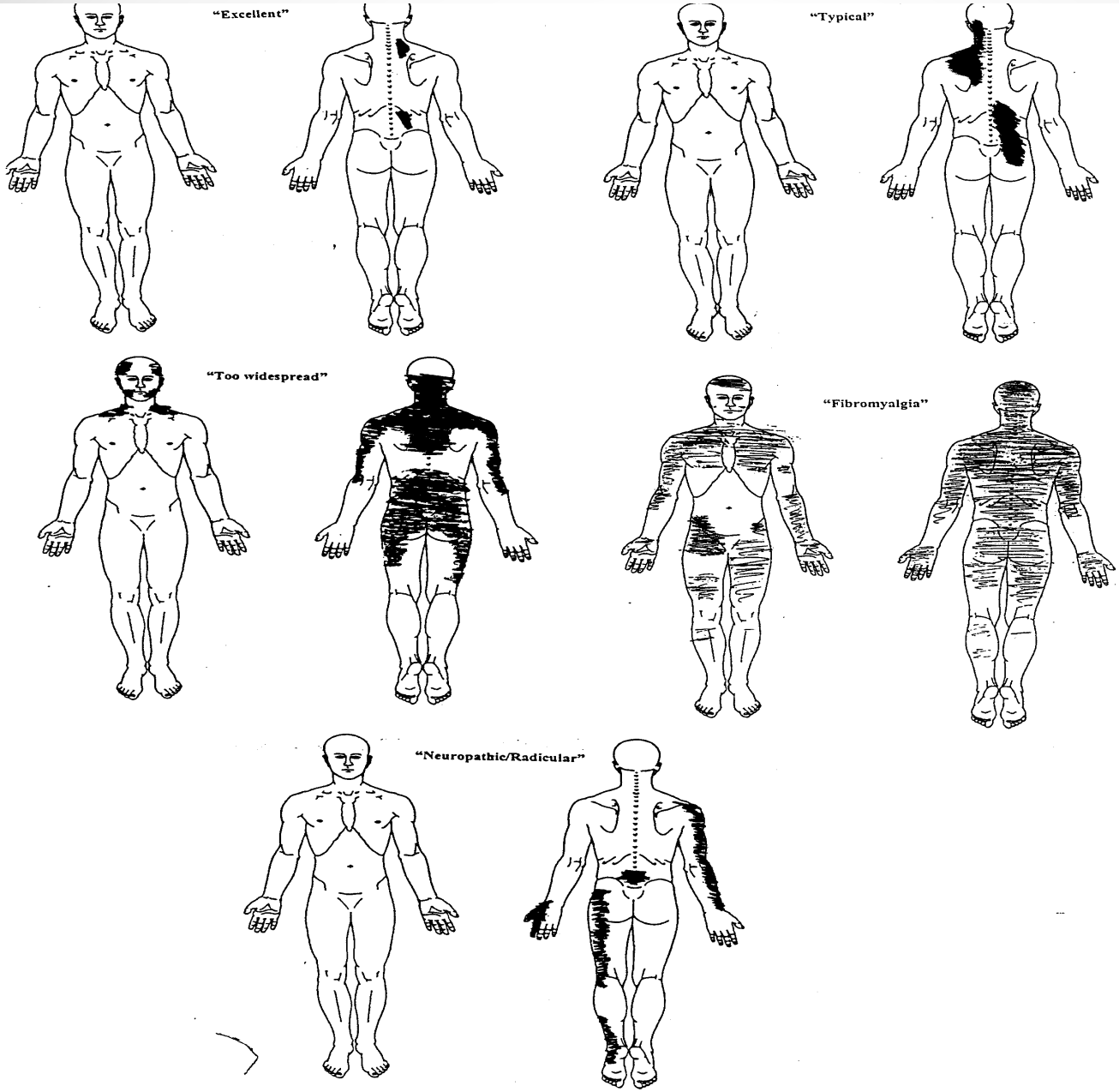
Treatment

Testing whether laughter *is* the best medicine

PAIN DRAWING

SHADE IN WITH A PENCIL ALL AREAS YOU HAVE PAIN.
(Don't forget to include the head or areas of lesser pain).
Use small x's to show any areas of numbness or tingling




"Excellent"
"Typical"
"Too widespread"
"Fibromyalgia"
"Neuropathic/Radicular"

DAVID R. HUBBARD, MD

SOURCE OF PAIN	DIAGNOSES	PHYSIOLOGY	SYMPTOMS	EXAM	TESTS	TREATMENT	PROGNOSIS
MUSCLE	STRAIN INJURY LOW BACK PAIN REPETITIVE TRAUMA "TMJ" TENSION HEADACHE MYOFASCIAL PAIN	MUSCLE STRETCH RECEPTOR OVER-STRETCH, AND ADRENALIN OVER-ACTIVITY	WIDESPREAD PAIN, STIFFNESS INCREASED WITH EXERTION, IMMOBILIZATION AND TENSION	TRIGGER POINTS ISOMETRIC CONTRACTION INCREASES PAIN	TRIGGER POINT EMG SHOWS SPASM LOCALIZED TO THE NIDUS OF THE TRIGGER POINT	GENTLE STRETCH BIOFEEDBACK EMG-GUIDED TRIGGER POINT INJECTION	RESOLVABLE BUT TENSION MUST BE ADDRESSED
NERVE COMPRESSION	RADICULOPATHY CARPAL TUNNEL DISC HERNIATION	COMPRESSION AND ISCHEMIA OF NERVE	RIBBONS OF PAIN, TINGLING AND NUMBNESS	STRETCHING NERVE INCREASES PAIN, DECREASED SENSATION IN NERVE DISTRIBUTION	MRI, CT OR MYELOGRAM SHOW COMPRESSION OF THE NERVE BY DISC, TUMOR OR OTHER TISSUE	SURGICAL DECOMPRESSION IF NERVE COMPRESSION PERSISTS	AFTER SURGERY, RISK OF RESIDUAL NERVE DAMAGE
NERVE DAMAGE	NEUROPATHY RADICULOPATHY	DEMYELINATION OR DISRUPTION OF NERVE	SAME AS NERVE COMPRESSION	DECREASED SENSATION IN NERVE DISTRIBUTION, LOSS OF REFLEXES	EMG-NVC SHOWS SLOWING OF NERVE CONDUCTION AND/OR TWITCHING OF MUSCLE	"ANTI-DEPRESSANTS" "ANTI-CONVULSANTS"	PERMANENT
NERVE TRACTION	THORACIC OUTLET SYNDROME PIRIFORMIS SYNDROME	STRETCHING OR CROWDING OF NERVE BUNDLES	TINGLING OF ENTIRE ARM OR LEG	PALPATION AND CONTRACTION OF MUSCLES INCREASES TINGLING.	EMG-NCV IS NORMAL	SAME AS FOR MUSCLE PAIN	RESOLVABLE ONCE MUSCLE PAIN RESOLVED
JOINT	OSTEOARTHRITIS DEGENERATIVE SPINE DISEASE RHEUMATOID ARTHRITIS	THINNING AND ROUGHENING OF JOINT SPACE	PAIN LOCALIZED TO JOINT	PASSIVE MOVEMENT OF JOINT INCREASES PAIN THICKENING OF JOINT	X-RAY SHOWS JOINT DEGENERATIVE CHANGES	NON-STEROIDAL ANTI-INFLAMMATORIES	SLOWLY PROGRESSIVE
MIGRAINE	MIGRAINE	SHUNTING OF BLOOD THROUGH THE MENINGES	ATTACKS OF PAIN, LASTING 1-3 DAYS, WITH NAUSEA	NORMAL EXAM	NONE	IMITREX	VARIABLE
"RSD"	COMPLEX REGIONAL PAIN SYNDROME	CONSTRICTION OF BLOOD FLOW IN THE HAND OR FOOT	PAIN IN THE HAND OR FOOT THAT DOES NOT FIT A NERVE PATTERN	SKIN TEXTURE AND TEMPERATURE CHANGES	SKIN TEMPERATURE MEASUREMENT	BIOFEEDBACK LIDOCAINE GIVES TEMPORARY RELIEF	VARIABLE
FIBROMYALGIA	FIBROMYALGIA	UNKNOWN	DIFFUSE PAIN, FATIGUE, INSOMNIA, DEPRESSION	DIFFUSE TENDERNESS IN ALL FOUR QUADRANTS OF BODY	NONE	ANTI-DEPRESSANTS NON-STEROIDAL ANTI-INFLAMMATORIES Sharp biofeed	SLOWLY PROGRESSIVE ? variable

Differential Diagnosis of FM and MPS

(adapted from Schneider, 1995)

Symptom	***Fibromyalgia***	***Myofascial Pain Syndrome***
Pain pattern	Bilateral & Widespread	Regional: Specific referred pain patterns
Morning fatigue	Yes	No
Sleep disorder	Yes: strong correlation with FM	Sometimes: secondary to pain & discomfort of MPS
Soft tissue findings	Tender point	Trigger point
Palpable changes	None	Distinct “nodularity” over TP; Palpable, taut “ropy” bands with associated features
Female/Male ratio	10-20:1	1:1

Differential (continued)

Symptoms	***Fibromyalgia***	***Myofascial Pain Syndrome***
History / presentation	Chronic, widespread pain; morning fatigue, stiffness and pain of unknown cause	History of acute or chronic muscle strain or injury; regionalized pain
Treatment approach	Treatment is systemic: Low dose anti-depressants Aerobic Exercise Psychotherapy Chiropractic manipulation	Treatment is specific and local: Accupressure Therapeutic stretch Injection

Treatment Considerations I

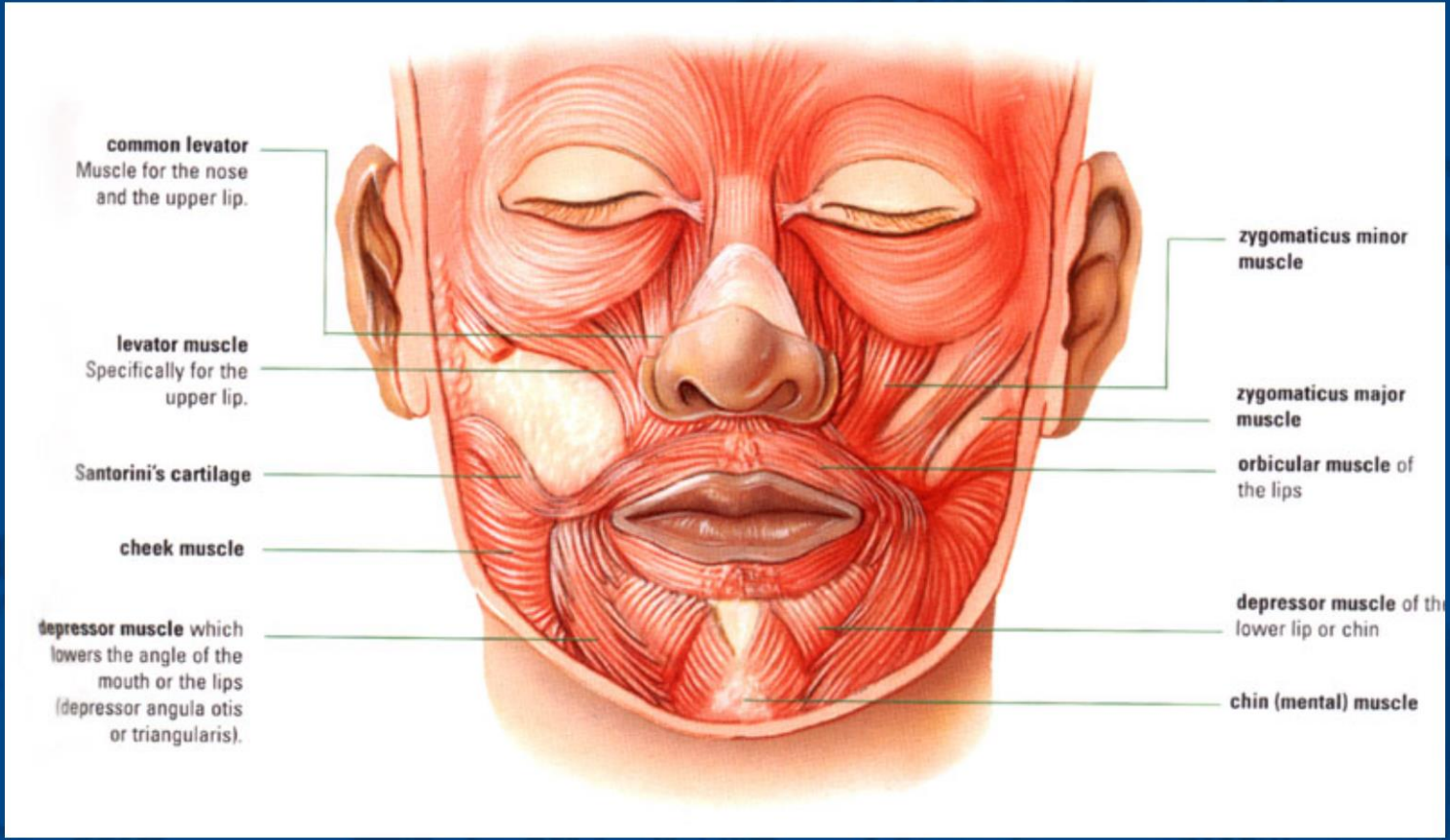
- Education/Attribution Shift
- Use grid, video, articles, persuasion, diagrams, etc. to achieve shift in causal attribution
- Physical management
  - Acupressure, acupuncture, theracane, tennis balls, passive stretches, moist heat, spray and stretch
  - Injections (Phenoxybenzamine, botulinum toxin type A, dry needling

Sharp Hospital Treatment Model I

- 1992 to present
- Average weekly census =225 patients (one of largest in world)
- 61% managed care, 33% workers' compensation
- 67% reduction in following year health care costs
- Despite average of 3.8 years disability, 67% returned to work

Parasympathetic "Accentuated Antagonism"

- "Vagal 'tone' predominates over sympathetic tone at rest. Under normal physiological conditions, abrupt parasympathetic stimulation will inhibit tonic sympathetic activation and its effects at rest and during exercise. This response is known as 'accentuated antagonism' "(Olshansky et al., 2011, p.863; Yang and Levy, 1984; Schwegler and Jacob, 1975; Levy, and Zieske, 1969)

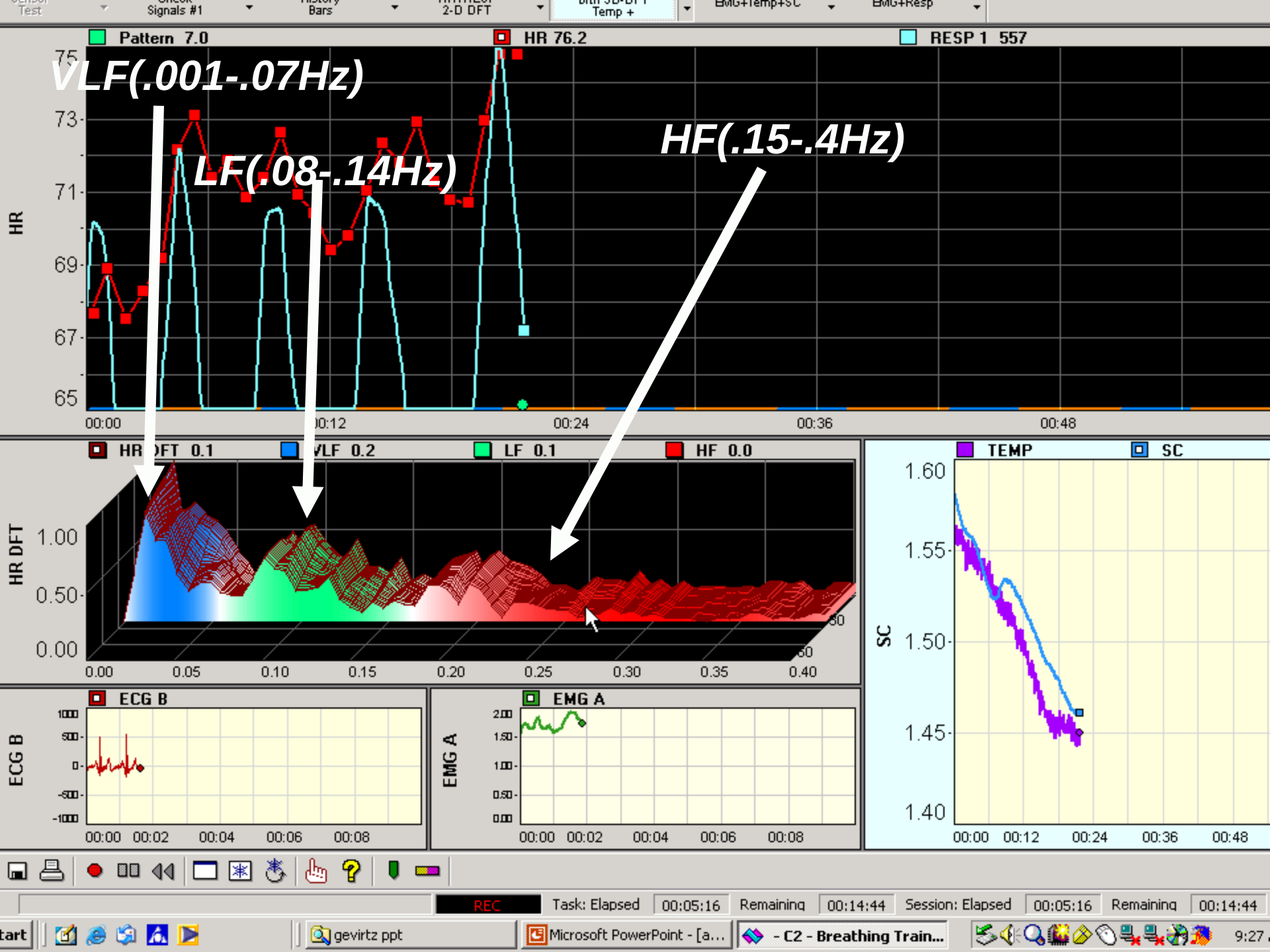

common levator
Muscle for the nose and the upper lip.
levator muscle
Specifically for the upper lip.
Santorini's cartilage
cheek muscle
depressor muscle which lowers the angle of the mouth or the lips (depressor angula otis or triangularis).
zygomaticus minor muscle
zygomaticus major muscle
orbicular muscle of the lips
depressor muscle of the lower lip or chin
chin (mental) muscle

Sharp Hospital Treatment Model II

- Muscle pain education/differential diagnosis
- Cognitive coping
- Physical coping/gentle stretching
- Medication management
- Use of interns
- De-emphasize traditional psychological models

Treatment II: sEmg Biofeedback Techniques

- Muscle awareness
  - Biofeedback, bilateral, symmetry, traps, frontal
- Frontal EMG
  - Cultivated Low Arousal
  - Facial Muscle Feedback
- Bi-lateral Trapezius Tx
  - For Bracing or Splinting
  - For Symmetry
  - For Breathing
- Specific Muscle Placement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HF(.15-.4Hz)
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EMG A
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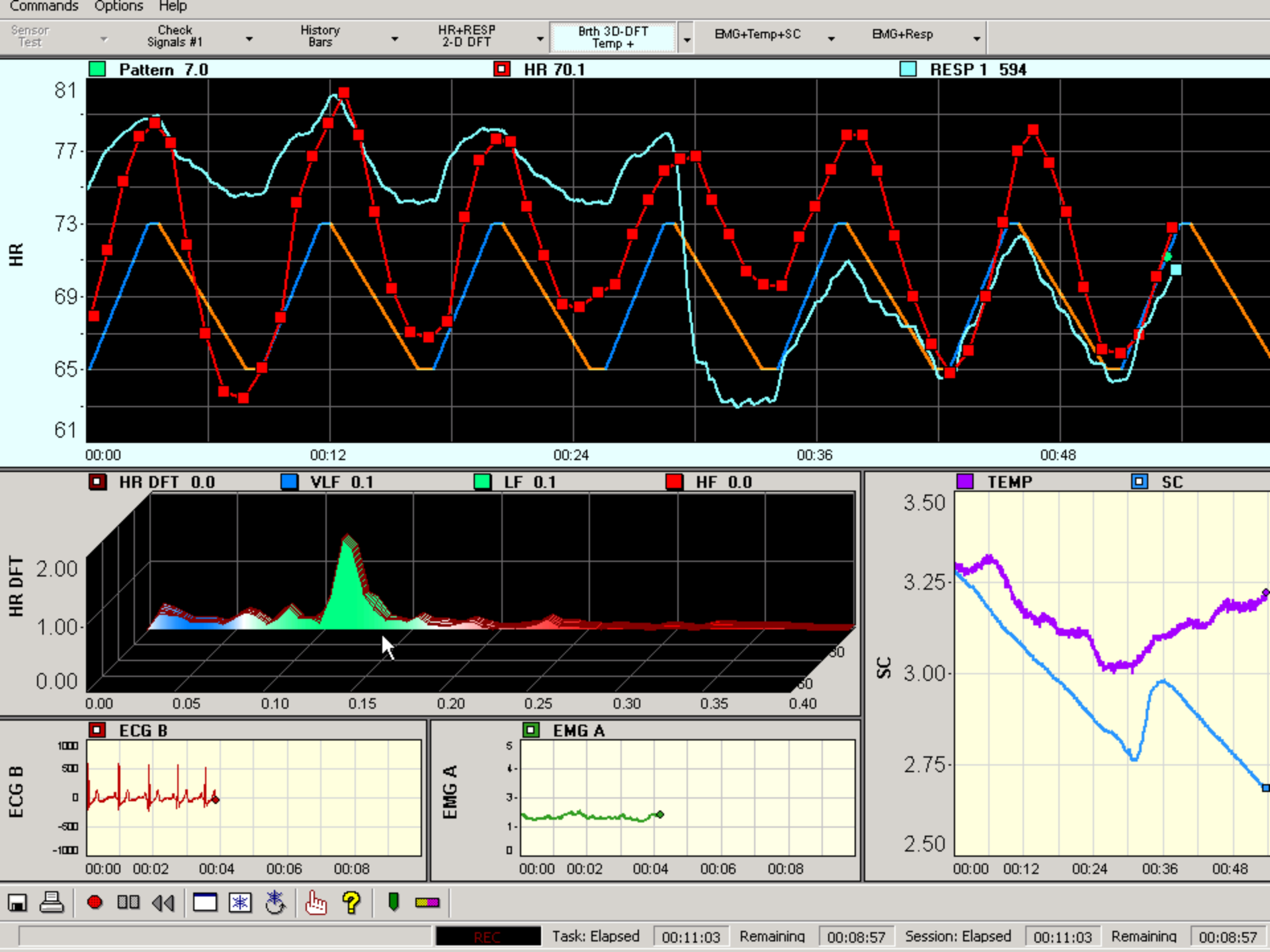
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BMG+Resp
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HR 70.1
RESP 1 594
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0.20
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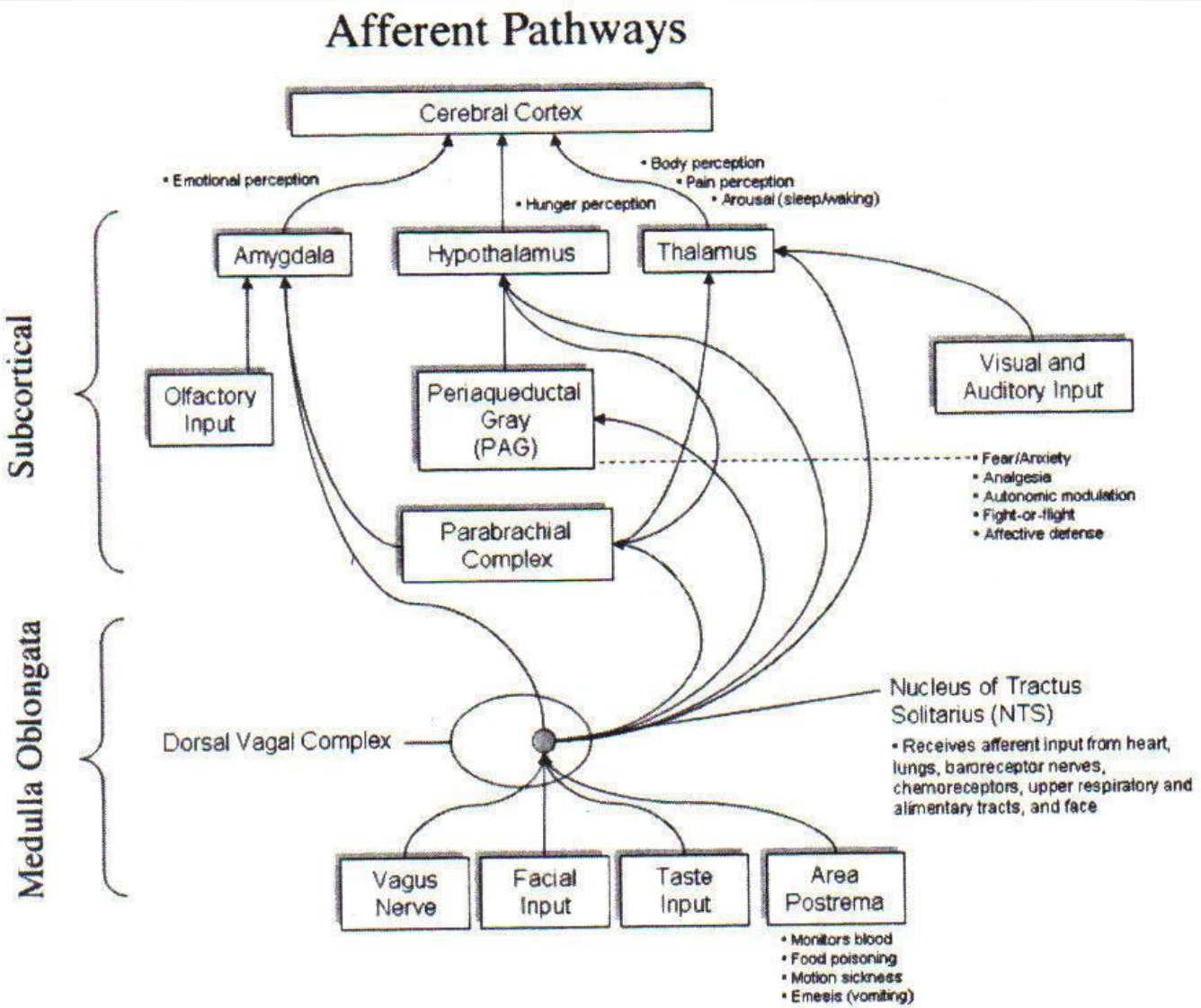
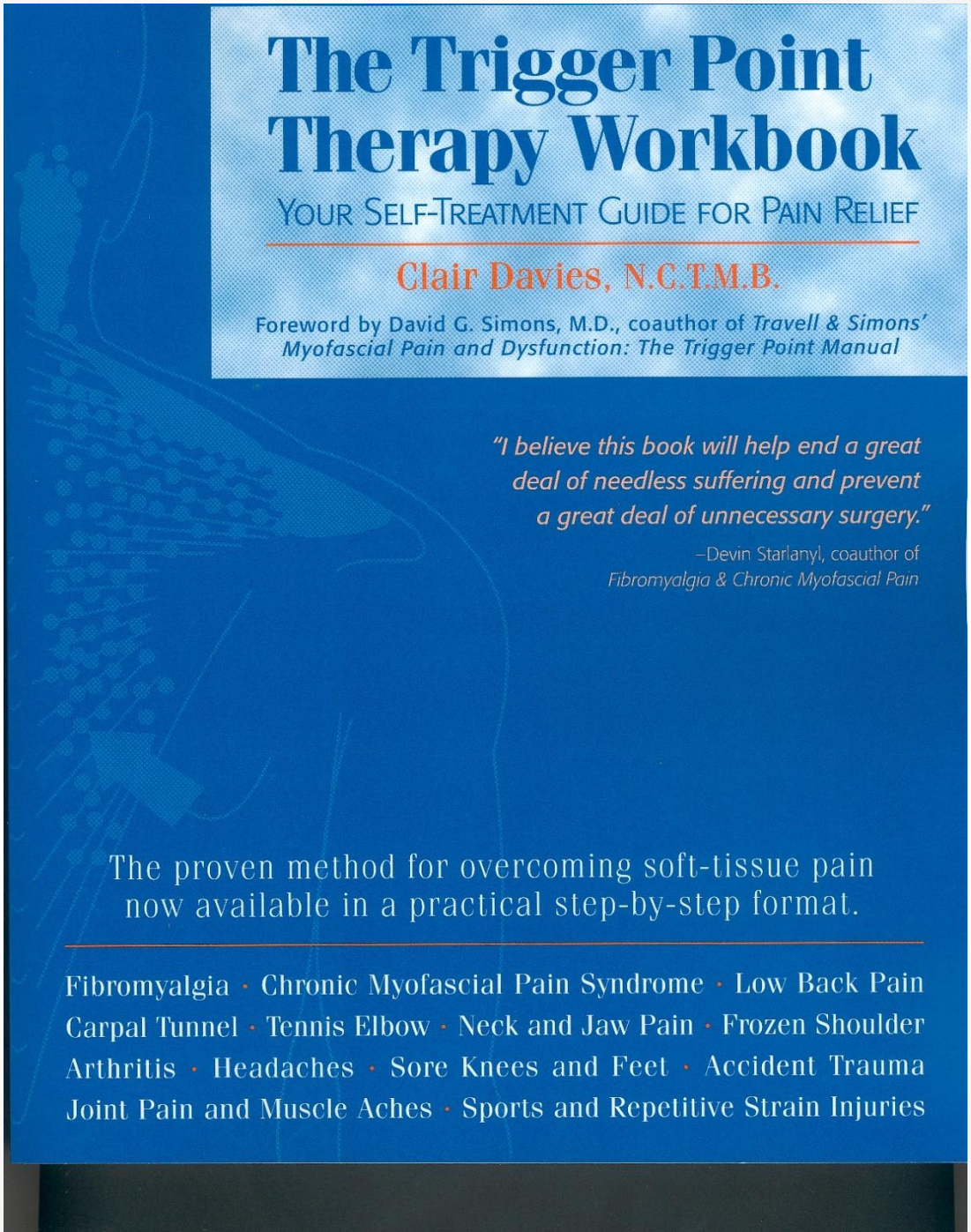


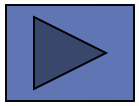
Figure 8. Afferent pathways. Diagram of the currently known afferent pathways by which information from the heart and cardiovascular system modulates brain activity. Note the direct connections from the NTS to the amygdala, hypothalamus, and thalamus. Although not shown, there is also evidence emerging of a pathway from the dorsal vagal complex that travels directly to the frontal cortex.

the familiar pattern, is what underlies the generation of sistent source of dynamic rhythmic patterns in the body.


The Trigger Point Therapy Workbook
Your Self-Treatment Guide for Pain Relief
Clair Davies, N.C.T.M.B.
Foreword by David G. Simons, M.D., coauthor of Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual
"I believe this book will help end a great deal of needless suffering and prevent a great deal of unnecessary surgery."
–Devin Starlanyl, coauthor of Fibromyalgia & Chronic Myofascial Pain
The proven method for overcoming soft-tissue pain now available in a practical step-by-step format.
Fibromyalgia · Chronic Myofascial Pain Syndrome · Low Back Pain
Carpal Tunnel · Tennis Elbow · Neck and Jaw Pain · Frozen Shoulder
Arthritis · Headaches · Sore Knees and Feet · Accident Trauma
Joint Pain and Muscle Aches · Sports and Repetitive Strain Injuries

Treatment Considerations III

- Breathing/relaxation
  - Cultivated low arousal, breathing retraining, mindfulness techniques, etc.
  - Resonant Frequency Training
- Problem solving
  - Using awareness of muscle tension, try to remedy causal situation(seeing the big picture)

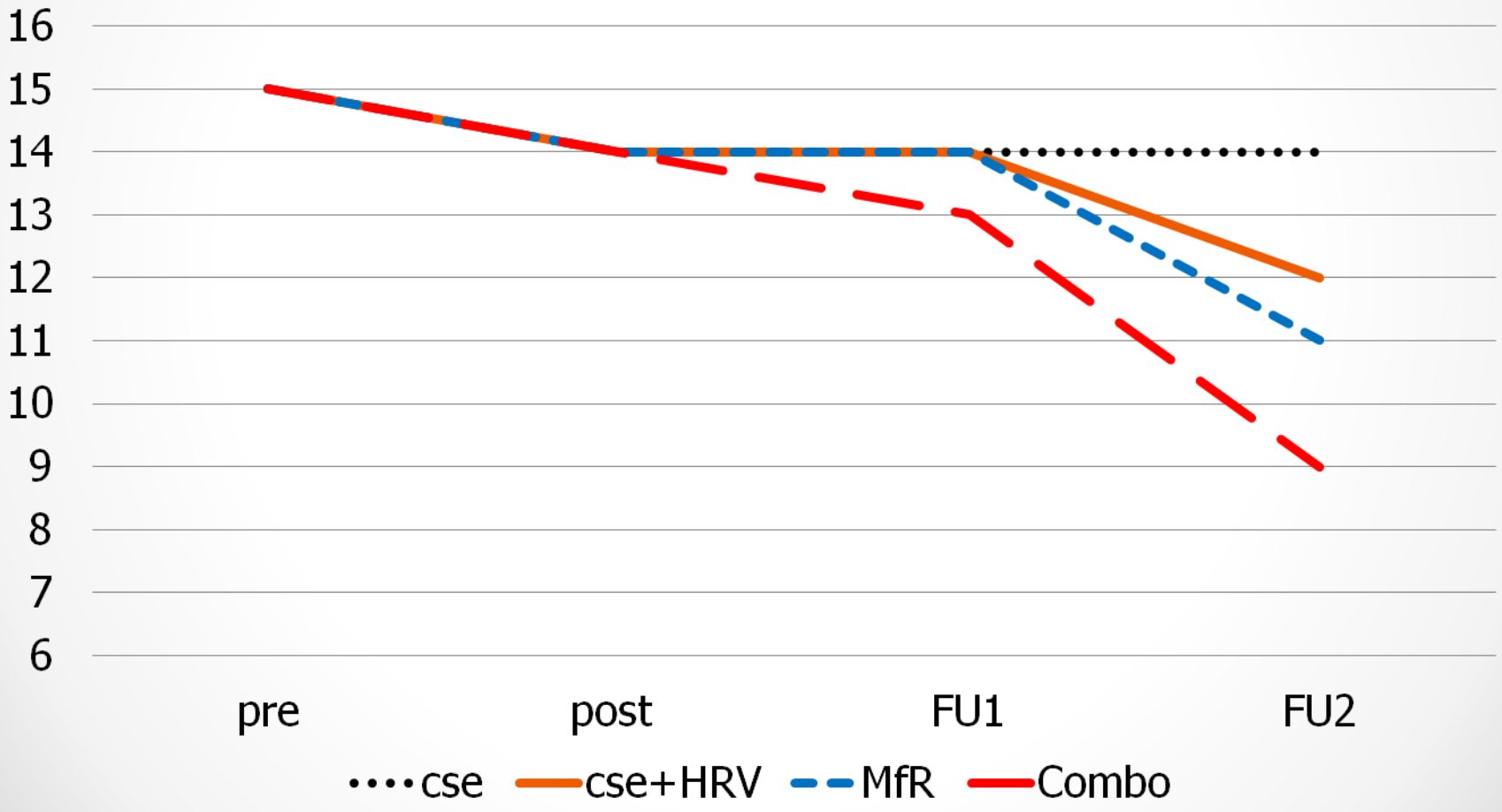


Treatment Considerations IV

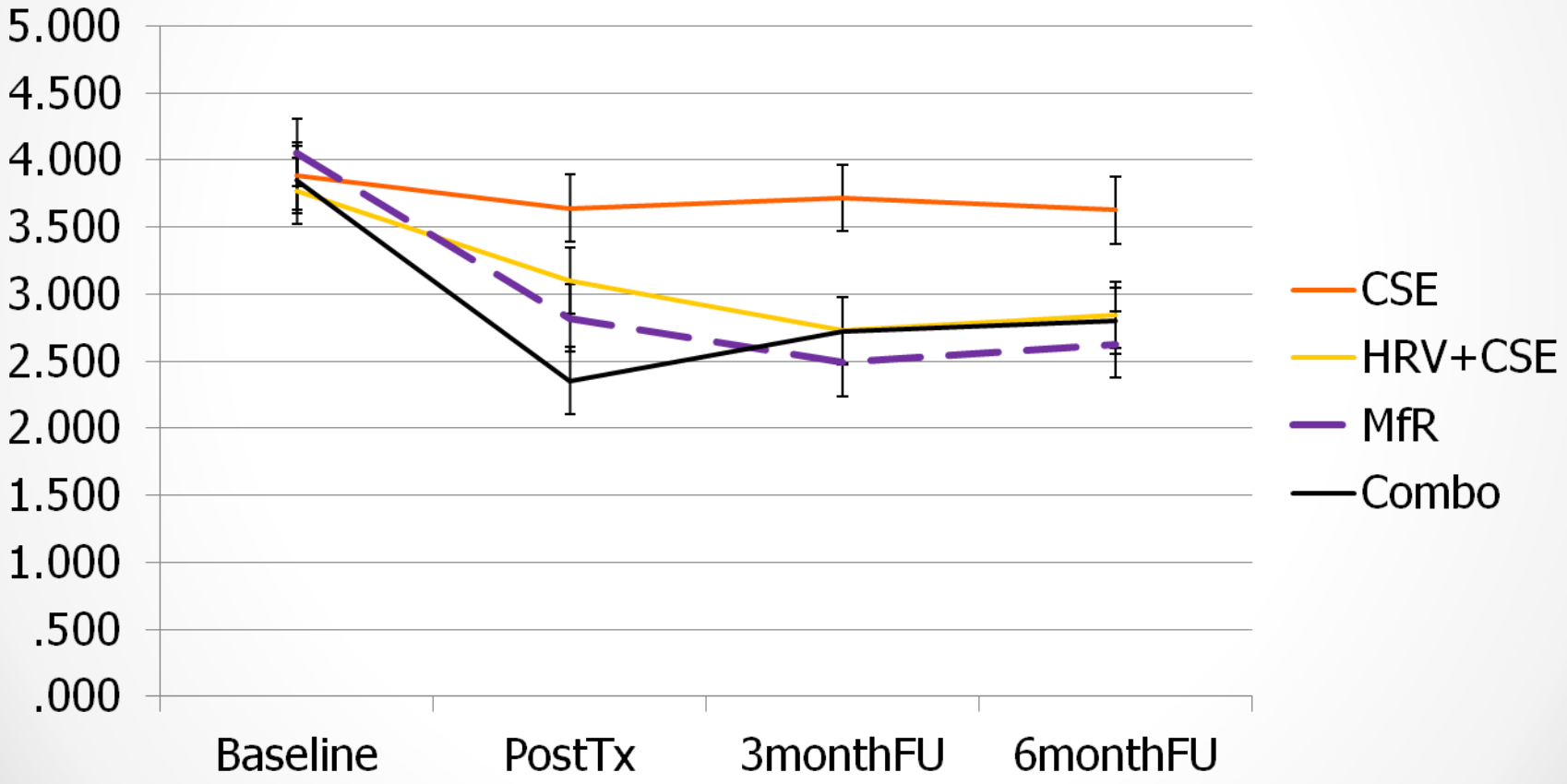
- Cognitive interventions
  - Since the model hypothesizes that persistent sympathetic activity (even if low level) stimulates activity in the TrPs, we now look for the "smoking cognitive gun". This is likely to center around distorted self-schema such as: "I'm only a valid person if I am pleasing others", or "If I am not perfect I am worthless".

Pain ratings across time

(Vagades, Gordon, Gevirtz, Andrasik (2013) N=124



Composite pain measure



Wrap-Up

- Summary of the arguments presented today
  - Epidemiology
  - Pathophysiology
  - Treatment
- Implications: Mind/Body techniques should be the first line treatment for muscle pain disorders